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PSYCHIATRIC ASPECTS OF EPIDEMIC ENCEPHALITIS*

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Since the earliest descriptions of epidemic encephalitis, most observers have recognized that some degree of psychic disturbance formed an essential feature of the clinical picture. Very few studies have, however, dealt particularly with the psychiatric aspects of this disease. In fact, a review of the extensive literature which has appeared during the past few years shows, in contrast to the intensive study and finer differentiation of the various neurologic syndromes, that the psychotic manifestations of encephalitis have on the whole received relatively little attention.

Those writers who have reported psychiatric observations have been concerned mainly with the initial or early mental symptoms, and remarkably little has so far been recorded as to the course and outcome of the mental disturbance; and the important questions of permanent mental damage and the more chronic psychotic manifestations have been discussed hardly at all. It is, of course, evident that a satisfactory solution of certain of the psychiatric problems presented by the disease will require a longer period for clinical observation of cases than has elapsed since the appearance of the epidemic in 1918-1919.

The material on which the present communication is based has been selected to meet, so far as possible, the requirements of a well-rounded psychiatric study. The group available for the analysis comprises eighteen cases. We have included only those about which there could be no doubt as to the diagnosis of lethargic encephalitis. Moreover, they are all cases about which we were able to get a fair anamnesis from relatives or friends. We were fortunate in being able to study cases in both general hospitals and state hospitals for the insane. We

* An abstract of this paper was read before the meeting of the Association for Research in Nervous and Mental Diseases, New York City, Dec. 28 and 29, 1920.

were thus enabled to compare early and late clinical pictures, and by looking up the patients discharged from the hospitals, we secured additional data on which to form an opinion as to the course and outcome of the disorder in each of the patients included in our series.

FREQUENCY OF SEVERE OR PROLONGED PSYCHOSES

It is probably true, as suggested by Barker,[1] that during the present epidemic many cases of encephalitis have been overlooked and undoubtedly many cases of psychoses are passing through the hospitals without their relation to encephalitis being suspected. We know of several cases of acute psychosis which were not recognized as an encephalitic disorder until the brain was studied postmortem.

In New York State most of the cases in adults with prolonged or severe psychoses eventually reach the state hospitals. The staff physicians of the institution have been, since the epidemic was recognized, on the alert for cases of the disease.[2] It is, therefore, of interest to learn how many cases have been received in the state hospitals. So far the number admitted because of grave psychoses is apparently small in comparison to the number of cases reported in the community and treated in general hospitals. During the hospital year from July 1, 1919, to June 30, 1920, in only twenty cases was the diagnosis of epidemic encephalitis considered probable among the 6,500 admissions to the New York state hospitals.[3]

CLINICAL GROUPINGS

The wide variety of terms used by different writers to designate the more striking psychotic manifestations of encephalitis indicates a lack of any well founded nosologic conception. The following list, by no means an exhaustive one, taken from recent articles, illustrates very well the wide range of designations applied to the mental disturbances accompanying or following epidemic encephalitis: acute delirium, maniacal delirium, afebrile mental confusion, amentia, mania, true mania, manic state, real hypomania, anxiety depression, excitable

1. Barker, Cross and Irwin: Epidemic Acute and Subacute Nonsuppurative Inflammations of the Nervous System, Am. J. M. Sc. **159**:157, 1920.

2. New York State Hospital Quarterly, November, 1920, contains an article on "Encephalitis Lethargica" by Drs. Montgomery and Waldo, of the Willard State Hospital and a report of five cases by Dr. Heyman of the Manhattan State Hospital.

3. Unfortunately, we have no data as to the number of encephalitis cases which developed in New York State during this period. We are informed by Dr. E. S. Godfrey of the state board of health that epidemic encephalitis was not made a reportable disease until June 1, 1920. During that month, the epidemic being already on the wane, only sixteen cases were reported in the state.

depression, melancholia, emotional stupor, hebephrenia, catatonia, lucid catatonic stupor, paranoid state, grave delusional psychosis, fully elaborated psychosis, epileptico-maniacal psychosis, Korsakoff's disease, hysterical attack and psychoneurosis.

It is unfortunate that psychiatric terms already identified with well-established clinical entities should have been so freely used even for descriptive purposes. Some observers, however, apparently do not use the terms merely in a descriptive way, as they seem to think that manic-depressive insanity, dementia praecox, paranoiac states and other constitutional psychoses may be definitely related etiologically to epidemic encephalitis.

Only a few observers have so far offered any systematic psychiatric grouping of the cases studied. Indeed, many writers have been content merely to mention and keep apart as "psychotic forms" or "mental forms" those cases in which the psychic manifestation overshadowed the neurologic syndrome, although admitting that in any of the various types, separated on anatomic grounds, a mental disturbance may exist. Some authors, as Buzzard, ignore the mental aspect of the disorder and adhere to a strictly anatomic classification. Others, as Tilney and Howe,[4] group their cases partly from the anatomic and partly from the psychiatric point of view. Hesnard[5] has perhaps attempted the most comprehensive grouping of psychotic syndromes. His main divisions are:

I. Mental disorders in the more common neurologic types. The mental disturbance, assuming a subordinate rôle, usually does not go beyond a state of lethargy or mild confusion.

II. Mental disorders in a group of cases in which the neurologic signs are less prominent while the psychotic disturbance stands in the foreground from the first. These are the "encephalitic psychopathies" for which the general designation of "acute psycho-encephalitis" is suggested.

As clinical varieties under the second division the author mentions the following:

1. Psychosomnolent form: Drowsiness succeeded by mild excitement (hypomania) passing into confusion which is later followed by depression.

2. Lucid catatonic stupor: General immobility, catatonia, preservation of mental clearness.

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4. Tilney, F., and Howe, H. S.: Epidemic Encephalitis, New York, Hoeber, 1920.

5. Hesnard: La Psycho-Encéphalite Aiguë Epidémique, l'Encéphale **15**:443 (July) 1920.

3. Acute delirious form: The delirium may be frankly hallucinatory, analagous to febrile delirium tremens.

4. Confusional form: Marked reduction of mental activity with clouding of the sensorium.

5. Korsakoff form: Disorientation with confabulation, often, but not always, accompanied by neuritic or poliomyelitic symptoms.

6. Miscellaneous psychic disturbances: Especially sequelae seen most often in children who show mental dulness or various changes in character and disposition.

An obvious objection to any such clinical grouping as the foregoing is that it tends to set up a false contrast between neurologic and psychotic syndromes. It arbitrarily leaves largely out of consideration the mental disturbances, often severe, which frequently accompany the more marked neurologic syndromes and tends to give the impression that when mental symptoms are pronounced the physical symptoms are likely to be slight and vice versa. We do not think there is any justification to speak of "psycho-encephalitis" and thereby suggest that the mental reaction in certain cases is more important than the physical aspects of the disease. A patient with mild mental symptoms at the beginning may later on show a marked psychotic disturbance and it is well known that the initial physical symptoms form no criteria of what the eventual neurologic syndrome may be.

Later we will show that the so-called "psychosomnolent form" and "lucid catatonic form" are not clinically distinct types. They are only in a very superficial way different from the other types, may occur during the course of any of the forms mentioned, all of which we find contain elements fundamentally more important in psychiatric differentiations than those given by Hesnard.

PSYCHIATRIC PROBLEMS OF EPIDEMIC ENCEPHALITIS

It is now known that epidemic encephalitis is an acute or subacute inflammatory disease of the central nervous system, a meningomyeloencephalitis. It is probable that the disease is caused by an infectious organism. Furthermore, it seems reasonably certain that as a result of the activity of the infective agent or its toxin within the body a varying degree of temporary or permanent damage to the nervous tissues takes place. As a consequence, psychic disturbances of varying degrees of intensity arise.

The psychiatric problems of epidemic encephalitis would, therefore, appear to lead directly to a consideration of those types of mental disturbance susceptible of being called forth by toxic-infectious causes which act deleteriously on the central nervous system. From the standpoint of clinical differentiation the first question might be: What are

the known characteristics of mental disorders arising on such an etiologic basis?

The studies of Meyer,[6] Bonhoeffer,[7] Hoch[8] and Bleuler[9] have shown that disturbance of mental function, like disturbances of other functional mechanisms, takes place in a relatively limited number of ways. Clinical studies have in fact made it possible to circumscribe a fairly small number of abnormal mental reaction types now recognized to be of fundamental importance in all questions of pathogenesis and symptomatology of mental disorders. So far as we can see at present, there are three mental reaction types which are of special psychiatric significance. These are:

1. *Organic Reactions.*—These are characterized essentially by impairment of apprehension, interference with elaboration of impressions, defects in orientation and retention; difficulty in activization of memories with variability in mental capacity and level of attention—the so-called mental tension defect. It is now well established that this reaction is seen only when the brain has been damaged, temporarily or permanently, including injury by toxic-infectious agents.

2. *Affective Reactions.*—These are characterized by emotional disturbances which follow essentially the pattern of the normal affective states. Typical examples are the manic-depressive oscillations and other benign emotional disorders.

3. *Trend Reactions.*—These are characterized by abnormal trends and ideas, an unusual attitude toward the outside world, with constitutional peculiarities and psychogenic mechanisms in the foreground. Examples are schizophrenic and paranoiac states.

While these mental reaction types are often encountered clinically in pure form, they need not coincide with what we call "mental diseases." In fact, one reaction does not necessarily exclude another. For instance, an affective or trend reaction may be superimposed on, or released by, an organic reaction, so that the resultant clinical picture is thereby given a special cast. Descriptive psychiatry has been, to a considerable extent, concerned with an effort to single out and identify as clinical entities groups of symptoms which belong to one or the other of these forms of mental reaction.

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6. Meyer: The Problem of Mental Reaction Types, Mental Causes and Diseases, Psychological Bull. **5**:245, 1908.

7. Bonhoeffer: Die Symptomatischen Psychosen in Gefolge Von Akuten Infektionen und Inneren Erkrankungen, Franz Deuticke, 1910.

8. Hoch: The Problem of Toxic-Infectious Psychoses, Bull. New York State Hospitals, November, 1912.

9. Bleuler: Dementia Praecox Oder Gruppe der Schizophrenien, Franz Deuticke, 1911.

It is our view, based on pathology and probable etiology, and also on clinical experience, that the psychiatric problem of epidemic encephalitis belongs essentially in the realm of the organic, more specifically the toxic-infectious mental reaction types. The present study is a clinical contribution in affirmation of this conception. Before proceeding to discuss our case material, we will sketch briefly the principal features of the organic mental syndromes.

The conception of an organic syndrome is not a new one in psychiatry, although the old and unsatisfactory designation of "organic psychoses" passed out of use as we became better able to differentiate various types of brain disease. A more precise description and designation of the organic reaction has resulted from the recent work of Kraepelin [10] and of Bonhoeffer [7] who have analyzed in detail the features common to all psychoses which develop in reaction to brain disease or injury. Bonhoeffer especially has extended our knowledge by studies of "exogenous" causes and the clinical pictures constituting the symptomatic psychosis, that is, those due to bodily diseases and poisons.

The *chronic* organic reactions are seen in arteriosclerosis, senile deterioration, chronic Korsakoff disease, paresis, brain tumor, etc., all of which show interference with mental grasp, diffuse memory defects, impairment of retention, variability in capacity and clearness, diminished ability for complicated thinking, and fluctuations in levels of attention—a typical mental tension defect.

The *acute* organic reactions have recently become better understood, chiefly as a result of Bonhoeffer's work on acute Korsakoff's disease, delirium tremens and other deliria accompanying physical diseases. The typical acute reactions are the deliria due to trauma, alcohol, uremia, lead and carbon monoxid poisoning, infections, bacterial toxins, etc. Brain torpor, somnolence or stupor may, however, be the most striking clinical expression of the reaction, replacing or alternating with the delirium. In the acute types, we encounter the difficulty in mental grasp and elaboration of impressions to the point of clouding of the sensorium, orientation and retention defects, and a striking variability in the level of consciousness: when lowered the train of thought becomes fragmentary (muttering and incoherence), when raised by questions and external stimuli the patient becomes more attentive. With clouding of consciousness, dreamlike ideas and hallucinations appear, and there is a marked tendency for habitual trends of thought and activities to determine the scene — the well-known occupation delirium.

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10. Kraepelin: Psychiatrie, Ed. 8, 1910.

Psychogenic deliria in contrast to organic deliria, as pointed out by Bleuler [9] and by Hoch,[8] present quite a different type of reaction. In them the reaction centers about the realization of a wish with more or less complete splitting off of the rest of the personality, excluding, so to speak, the outside world or falsification of reality in harmony with the main trend.

As already intimated, even in the purest organic reactions, it is necessary to consider various modifying elements, particularly the personality make-up, the special etiologic factor at work, the intensity and duration of its action and predilection for definite parts of the brain. The widespread cropping out of constitutional traits and the appearance of various affective and trend reactions in the organic psychoses need not be dwelt on at present.

In concluding this part of the discussion we will simply mention the more common clinical forms of the acute organic reactions as they occur in psychiatric practice. Later we will discuss these types in relation to the symptom-pictures encountered in encephalitis.

1. Acute delirium: the organic features of which are plainly evident.

2. States of psychic torpor: various grades of mental dulness, somnolence, stupor or coma.

3. Amnestic-confabulatory complex: acute Korsakoff syndrome.

4. A group of more complicated psychotic reactions to which the term amentia [11] is often applied. In these the organic elements, disorientation and clouding of the sensorium, are combined with emotional disorders, trends and psychomotor disturbances which are not usually so prominent in the other acute organic syndromes. In these more complex cases, the personality traits and various psychogenic reactions come more to the front and thereby give a special cast to the clinical picture.

GENERAL SURVEY OF CASE MATERIAL

In Table 1 are presented the principal features of the neurologic and psychotic syndromes as encountered in our eighteen cases.

By far the most constant physical symptom in this series of cases was disturbance in the oculomotor mechanisms alone or in combination with other physical signs. This was plainly evident in all cases except one which was a pseudoparkinsonian type (Case 6).

Next in frequency among physical manifestations were pseudoparkinsonian symptoms, noted in six patients (Cases 3, 4, 5, 6, 7 and 11).

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11. The term "amentia" is not free from objection, but it is a useful designation in the sense here indicated for a clinical group seen in psychiatric hospital practice.

In two cases, there were symptoms of a transitory nature pointing to the cerebellum (Cases 9 and 11).

In two patients, a thalamic type of facial weakness was noted and in one of these there was uncontrollable laughter (Cases 5 and 12).

In one patient, meningeal symptoms were prominent at the beginning (Case 15).

In one patient, a lasting hemiplegia developed during a prolonged stupor (Case 17).

In eight cases, the physical symptoms have cleared up entirely, leaving ten cases in which there are light or severe physical residuals.

In general, we were not able to make any correlation between types of physical symptoms and the form or outcome of the mental disturbance. It is, however, interesting to note that of the ten patients with physical residuals not one can be said to have recovered completely mentally. On the other hand, of the eight patients without physical residuals, two have fully recovered mentally.

A general survey of the psychotic syndromes reveals that in every case there was at some time during the clinical course a period of pathologic drowsiness or mental torpor varying in degree from abnormal sleepiness to stupor or coma. The fact that this symptom appeared in all of our patients tends to make us think that cases in which it does not occur at some time during the course of the disease are the exception. Its appearance is very irregular. It may constitute the chief feature of the mental picture or it may appear for a brief period only during the course of other mental disturbances. Thus, we find it as an initial symptom, or following a delirium, excitement or period of insomnia, or it may immediately precede the convalescence.

The next most striking feature of the psychotic picture was the appearance of delirium or mental confusion. This symptom occurred with varying intensity and duration in fifteen of our eighteen cases. In the three cases in which it was not apparent, we do not, however, feel that delirious features in the sense of a temporary alteration in the level of consciousness with impairment of mental grasp, etc., can be definitely excluded. These three patients, unfortunately, could not be sufficiently examined for the lighter grades of impairment of the sensorium and delirious "dips" (Cases 1, 4 and 5). No patients presenting an acute or chronic Korsakoff mental picture have been observed by us.

A point of special interest is the appearance of trend reactions and emotional disturbances outside a setting of delirium or mental confusion. In general, the persistence of a delusional trend after the sensorium has cleared is a rare occurrence and must be considered in relation to certain constitutional factors which will be discussed later.

TABLE 1.—PRINCIPAL FEATURES OF THE NEUROLOGIC AND PSYCHOTIC SYNDROMES ENCOUNTERED IN EIGHTEEN CASES

Case No.	Neurologic Syndrome	Psychotic Syndrome
1	Oculomotor with choreiform twitchings; paresthesia, left hand and involuntary movements after nine months	Insomnia, mental and motor hyperactivity of occupational type, followed by drowsiness; long period of physical and mental insufficiency, gradual improvement; after nine months full recovery (?)
2	Oculomotor; Benedikt's syndrome; residual tremors, left arm	Drowsiness; slight delirium and nocturnal restlessness, then lethargy; no residuals after six months except possibility of slight change in disposition
3	Oculomotor; Pseudoparkinsonian; no residuals	Lethargy; semistupor; disturbance in time relations; change in disposition; subjective impairment in powers of memory after seven months
4	Oculomotor; partial left hemiplegia; pseudoparkinsonian; residual slight rigidity of upper extremities	Vague fears; drowsiness; lethargy; alteration in character apparent after one year
5	Oculomotor; severe pseudoparkinsonian; thalamic facial weakness (left); after one year residual parkinsonian features	Stupor, mutism and tendency to maintain given positions; then euphoria and uncontrollable laughter; no mental residuals after one year except possibly abnormal stubbornness
6	Pseudoparkinsonian; general rigidity, mask-face, unilateral pains in limbs; no residuals	Lethargy; confusion, prolonged immobile state and gazing; tearful on stimulation; anxious ideas and a few hallucinations; recovery in ten months, but possibly more emotional than formerly
7	Myelo-encephalitis (cervical); oculomotor; pseudoparkinsonian; residual weakness and atrophy of muscles of shoulder girdle	Lethargy; delirious episode; anxious depressive mood; after eight months residual nonincapacitating depression
8	Oculomotor; occasional blurring of vision after eight months	Nocturnal insomnia with occupational delirious episodes; somnolent during the day; rapid improvement but persistence after eight months of somnolent tendency
9	Oculomotor with suggestion of cerebellar involvement, dizziness and disturbance in equilibrium; no residuals	Insomnia, hallucinatory excitement, then short, violent delirium; drowsy period and recovery in two weeks; no residuals after eight months
10	Oculomotor; slight thalamic facial weakness (left); tremors in hands; no residuals	Insomnia, then confusion, delirious ideas and fear followed by lethargy; change in disposition apparent after ten months
11	Oculomotor with suggestion of cerebellar involvement, later involuntary movements, facial weakness and parkinsonian gait; residual squint	Acute hallucinatory delirium interrupted by stupor; then a weak, irritable state followed by hyperkinetic reaction and elation with clear sensorium; no defects in memory or mental capacity; excitement continues after one year.
12	Oculomotor; tremors; pyramidal tract involvement (right); residual speech and facial tremor	Stupor and delirious excitement, then euphoria and uncontrollable laughing attacks; after six months shows poor emotional control; episodes of anger and crying
13	Oculomotor with muscular twitchings in limbs; transitory Argyll Robertson pupil; no residuals	Delirium followed by lethargy; peculiar emotional reactions, inertia and low mental tension but no definite loss of mental capacity; unchanged after eleven months
14	Abrupt onset head pain; unilateral convulsion then coma; slight facial weakness; no residuals	Coma; afterward disorientation and confusion with dreamlike ideas and hallucinations; some restlessness and emotion; change apparent after three months
15	Oculomotor with meningeal symptoms; no residuals	Initial somnolence followed by hallucinatory delirium; recovery in three months with amnesic period; no residuals after one and one-half years
16	Oculomotor with left facial weakness, leg pains and sensation of twitching in tongue; physical symptoms persist after two years	Stupor, then depressive complaining attitude in reaction to physical condition; paranoid episode with hallucinations; trend not further elaborated after two years
17	Oculomotor with hemiplegia developing during stupor; residual hemiplegia	Stupor with confusion followed by suicidal depression, hallucinations and fears; peculiar emotional reactions; no definite mental deterioration; psychosis continues after fourteen months
18	Oculomotor; tremor of hands and tongue; no residuals	Stupor followed by occupational delirium; schizophrenia in individual with former tendencies in that direction; unchanged after ten months

On the other hand, we have found in a large majority of our cases peculiar emotional states which have persisted after all other mental symptoms have disappeared, so that a special type of residual seems to occur. Severe or prolonged psychotic manifestations occurred in four cases: one patient, after the stupor, developed a prolonged suicidal depression (Case 17); another, also after a stupor, developed a depressive paranoid state (Case 16); another one, after a severe delirium, showed a prolonged hyperkinetic condition with elation or irascibility (Case 11)); and in one patient a latent schizophrenia was brought to the surface with subsequent development of very marked symptoms (Case 18).

SLEEP DISTURBANCE

In fever, even occasionally in a mild febrile state, there is often undue drowsiness. In numerous organic brain conditions, for example, tumor, diffuse exudative disease, hemorrhage, to name only a few, as well as in states of so-called toxicosis resulting from metabolic disorders, there are disturbances of various degrees in the same field. It is apparent that pathologic sleep has a wide occurrence in neuropsychiatric conditions. In encephalitis such a disturbance is the most obvious mental alteration. At the beginning of the discussion, this disturbance is best put in terms of hyperfunction and hypofunction, for in this way it will be quickly brought to mind that not all patients with encephalitis show somnolence. Hypofunction of the sleep mechanism is frequently present instead.

On the hyperfunction side the excess of sleep in its mildest form amounts to a mere drowsiness. This stands at one end of the hypersomnic scale with coma at the opposite. It is serviceable to recognize the four gradations of drowsiness, lethargy, stupor and coma. In this way certain clinical distinctions are expressed and individual states more accurately described. The differences are those of degree.

Regarding the drowsy state the term defines itself. In it a certain more than usual desire to sleep and a tendency to doze does not noticeably upset the person's regimen.

When lethargy sets in, sleep of a special kind occurs. The patient sleeps more than is normal, possibly both day and night; his former occupations are necessarily curtailed. When asleep he can be very readily awakened completely. He may not return to sleep immediately, is not necessarily prostrate and may make voluntary efforts to keep busy at something. One of our patients, over a six weeks period when there was great lethargy, forced herself, under a physician's advice, to take long daily walks. This patient remained clear in her sensorium, except for a difficulty with the facts of time, indicating the kind of clouding apt to accompany that degree of sleep disturbance.

In stupor the patient is of necessity in bed and initiates nothing. There is a great reduction of activity. The patient is difficult to arouse, there is incomplete correlation between stimuli and reaction, and grave disturbance of the sensorium may be present. This may be sufficient to produce complete disorientation.

The patient in stupor may not always keep the eyes closed. The patients in Cases 5 and 6 went through an immobile state with mask-like facies, mutism and open eyes. Their difference in this may invalidate such cases for discussion under the general heading of sleep disturbance. One patient (Case 5) was not asleep, and in retrospect, he says that he understood everything and that he was unable to talk because of the rigidity of his tongue, lips and all of his vocal apparatus. These cases illustrate that the sleep disturbance so-called is only partly an alteration in the level of consciousness and that with it there is an activity disturbance. We do not think we have enough insight into gradations between variations in activity and somnolent states to warrant arbitrary limits in defining terms.

An all embracing inertness with the apparent suppression of reactions to virtually all external stimuli marks the fourth and most marked gradation, coma. Some of the writers who have used such descriptive terms as "twilight state" and "sleep of closed eyes" have plainly had in mind only the phase which is called lethargy in our nomenclature.

It has been suggested that the ease with which a lethargic patient can be aroused and the brightness and alertness then often shown are peculiar to encephalitis. It must be admitted that such a reaction is not usual in toxic-organic somnolent states, although something like it may be seen in brain tumor cases with facetiousness. On the other hand, in encephalitis it is often a question of degree or depth of the disturbance, as demonstrated by the stuporous or comatose patients who cannot be aroused, or if they can be, they show plainly dulness, lack of clearness or confusion.

When the sleep function is diminished or suppressed instead of augmented, the result is a hyposomnia, more commonly called insomnia. Climenko [12] has found insomnia as a symptom of onset. We have a similar history in three of our cases. Abrahamson [13] describes hypersomnia as an onset reaction and this occurs without doubt in the great majority of cases. Of the hypersomnic states, drowsiness, lethargy and stupor are frequent. Coma, in contrast, is not usual. It occurs in two settings. In one, the coma develops out of the stupor and marks an

12. Climenko: Encephalitis Lethargica, New York M. J. **111**:531 (March 27) 1920.

13. Abrahamson: Mental Disturbances in Lethargic Encephalitis, J. Nerv. & Ment. Dis. **52**:193 (Sept.) 1920.

intensification of the latter, coincident with an alarming advancement of the disease in its general features. Coma in this instance is almost always terminal. The other setting in which we have found coma is distinctly opposite in nature; namely, the coma has come on at the very onset of the disease. In one instance, it came with lightning rapidity upon an individual who until that time had been in health (Case 14). A convulsion inaugurated the disease and established the coma. A case with similar onset is described by Guillain.[14] Both of these patients progressed to recovery—one with a certain alteration of character. Coma, therefore, occurring at the onset gives quite a different prognosis from that developing later in the course of the disease.

Though as mentioned before, the insomnic picture has been noted by some as a preliminary onset symptom, in the majority of cases it develops later, if at all. In these instances, hypersomnia by a gradual transition gives place to insomnia. Bremer [15] says that the insomnia so frequent at the end of encephalitis is sometimes accompanied by an intense psychic overactivity. He mentions the case of a physician who had had the illness and had experienced the insomnia and hyperactive features. This physician reported that at first the insomnia was almost agreeable because of the lucidity and power of memory evocation which characterized it. Something much resembling it occurred in our Case 1.

The hypersomnia and the hyposomnia may alternate in the same patient through a definite day and night cycle, with a reversal, however, of the usual order. For example, such a patient, instead of sleeping at night, shows great restlessness and only with the coming of morning is able to sleep. The sleep which then ensues is the gripping lethargy which it is easy to interrupt but not possible to terminate. This rhythmicity in reverse order of the normal has been most frequently seen in children. Six such cases, all children, are described by Happ and Blackfan,[16] who attach a diagnostic significance to this sequence. In one of our adult patients (Case 8), this day and night cycle is recorded.

It is doubtful if speculation is profitable regarding the causation of these alterations of sleep. The question of morbid sleep has always dealt with a multiplicity of factors. Regarding normal sleep, Dana [17] writes, "Sleep is a biologic phenomenon which needs to be explained

14. Guillain: Encéphalite Lethargique Avec Crise Epileptique Initiale, Bull. méd. **34**:983 (Nov. 6) 1920.

15. Bremer: Formes Mentales de L'encéphalie Epidémique, l'Encéphale **15**: 517 (Aug.) 1920.

16. Happ and Blackfan: Insomnia Following Acute Epidemic Encephalitis in Children, J. A. M. A. **75**:1337 (Nov. 13) 1920.

17. Dana: Morbid Somnolence and Its Relation to the Endocrine Glands, Med. Rec. **89**:1 (Jan. 1) 1916.

only as the waking state or the diastole and systole of the heart need to be explained. It is not forced upon the system by any special hypnotizing secretion. It is part of the inherent and rhythmic habit of living tissue." Dana points out that in sleep there is a blocking of the sensory inflow and of many association paths, and that this nerve block is caused ordinarily and in part by fatigue products acting on the synapses of the nerve cells. He follows the suggestion of Gemelli that pituitary secretion perhaps has the power of antagonizing the blocking effect of these products. If pituitary secretion is lessened, the blocking action of fatigue products is greater, and morbid somnolence occurs. Cushing and Goetsch [18] had previously compared the physiology of the hibernating animal with the drowsiness and metabolic phenomena in hypopituitarism.

Inasmuch as the lesions of epidemic encephalitis so frequently focus about the cerebral peduncles with partial blocking of the aqueduct of Sylvius, the development in these cases of internal hydrocephalus can hardly be rare as pointed out by Buzzard.[19] Dunlap [20] has found in localized midbrain edema pathologic evidence of this occlusion. The bearing of this apparently frequent pathologic condition upon the question of the somnic alteration of the patient may depend upon an association between hydrocephalus and hypopituitarism as suggested by Pollock.[21] This is in line with previous analogous observations of Marinesco and Goldstein,[22] of Bailey and Jelliffe,[23] of Cushing [24] and others. This association would appear to rest upon the interference with the exit from the ventricles of pituitary secretions which normally reach the subarachnoid spaces and there come in contact with the cerebral cortex.

If we follow this suggestion, we are led to account for somnolence as a pressure effect of hydrocephalus upon the pituitary gland. But hydrocephalus causes pressure elsewhere, for example, on both thalami and on the cortex as well. Even presuming that hydrocephalus is a necessary step in the development of somnolence in these cases, it

18. Cushing and Goetsch: Hibernation and the Pituitary Gland, J. Exper. Med. **22**:25, 1915.

19. Buzzard and Greenfield: Lethargic Encephalitis, Brain **42**:305, 1919.

20. Dunlap: To be published.

21. Pollock, L. J.: Hypopituitarism in Chronic Hydrocephalus, J. A. M. A. **64**:395 (Jan. 30) 1915.

22. Marinesco and Goldstein: Two Cases of Hydrocephalus with Generalized Adiposity, Nouv. iconog. de la Salpêtrière **22**:628, 1909.

23. Bailey and Jelliffe: Tumors of the Pineal Body, Arch. Int. Med. **8**:851 (Dec.) 1911.

24. Cushing: The Pituitary Body and Its Disorders, Philadelphia, J. B. Lippincott Company, 1912, p. 202.

is not clear in what manner it works—whether through effects on the pituitary gland or through pressure on the thalami, cortex or other areas. Kennedy [25] believes that pathologic changes based on localization of the virus in the thalami or anatomic interruption of afferent paths to the thalami explain the somnolence. Others explain it in terms of changes at the synapses of the cells, presumably of the cortex. To speak again of the pituitary gland, specific alterations in that gland distinct from secondary pressure effects may be the factor to cause somnolence. Of all these theories, none can be said to be conclusive. Perhaps the chief argument against a localized thalamic lesion being the cause of the somnolence is the type of case which shows a twenty-four-hour cycle, stupor during the day, and restlessness, often extreme, during the night.

The hyposomnia and restlessness are just as little understood as the somnolence. It would seem that endocrinal factors would most readily permit of alternating combinations of the hypersomnia and hyposomnia and of strange day and night cycles.

DELIRIUM

It is the generally accepted view that delirium, if the psychogenic variety is excluded, points to a brain condition, an injury in the broadest sense, of a transient or permanent nature. The result is an interference with mental function the clinical expression of which is the syndome of delirium. We have already drawn attention to the resemblance of the delirium of trauma, alcohol, fever, etc., to the well-known organic types of mental disturbance and have shown the justification for regarding delirium as the acute form of the organic mental reaction.

The delirium of encephalitis presents the general characteristics of the acute organic disturbance. It occurred in fifteen of our eighteen cases in varying degrees of severity. We do not think that the three cases in which delirium was not recorded can be said to have been carefully enough observed or examined for one to say that mild transitory delirium did not occur. Delirium may appear at any time during the course of the disease. It is usually not prolonged and may in fact be limited to fleeting delirious episodes, especially at night. The cycle of nocturnal delirium and diurnal somnolence is often seen (Cases 8 and 12). One patient retrospectively remarked, "I was out of my head at night, but sensible during the day."

The most frequent sequence in our cases was for an initial drowsy or stuporous state to be followed or interrupted by delirium. Transient delirious features during a stupor may be easily overlooked. The

25. Kennedy: Epidemic Encephalitis with Stupor, Med. Rec. **95**:631, 1919.

patient may mutter only for a brief period, pick at the bed clothes, be harder to arouse, or the attention be more difficult to fix than usual.

In all of our cases beginning with insomnia, there followed a delirious phase which in turn was succeeded by drowsiness (Cases 9 and 10).

The content of the delirium of epidemic encephalitis, as in organic deliria generally, tends to center about habitual trains of thought and the usual daily activities of the patient, while combined hallucinations and illusionary falsification of things in the immediate environment are prominent. One patient, a former sailor, got on the roof at night and tried to climb the chimney imagining he was putting up a rigging. Another, a stevedore, imagined he was back at his work on the docks. A school teacher acted and talked as if she were in the class room with her pupils.

In some instances, the delirious content was apparently determined by disagreeable somatic sensations. One patient (Case 7) with a cervical involvement and root pains talked in a delirious way about having three arms, and at night said to his wife, "Wipe the blood off my arm—that other arm." Another patient (Case 13) who complained a great deal in the beginning of general soreness, pains and heat sensations, talked in his delirium of red pepper in the bed, of the devil's powder burning him all over, of feeling electric shocks, etc. Another patient who had a cystitis and required catheterization talked a great deal in her delirium of a criminal abortion.

In some cases, approaching more the amentia type of reaction, the delirious content is less simple, there being more evidence of complex trends. One patient (Case 14) confused her husband with her brother and said, "I am married twice, I can't think of the other man but I have two children." She wanted to be allowed to have "two eggs" in bed with her. Once she threw off the covers and called out that the eggs (imaginary) were not touching her, that nobody was in bed with her.

It is usually found that amnesia covers more or less the delirious period as is the case in alcohol and other toxic-organic deliria. As in the latter so in encephalitis, the recollection of the delirious trains of thought may be quite well preserved, while there is partial or complete amnesia for external events. The completeness of the amnesia for happenings in the environment varies, of course, with the depth of the delirium and is apt to be "patchy," in harmony with the fluctuations of the level of consciousness. By way of contrast, it is of interest to note in this connection that in the typical psychogenic deliria, as seen for instance in hysteria, the content is usually completely suppressed, the patient having on recovery no recollection of the delirious trend.

EMOTIONAL REACTIONS

The mood alterations which accompany encephalitis have, because of their prominence and variety, always attracted a great deal of attention. Some writers have emphasized the lack of a normal emotional responsiveness, ranging from mild indifference and slight dulling to profound apathy and absence of all affective reactions. Kennedy and others have described as one of the most characteristic symptoms the "emotional stupor," in which condition the patients are totally unable to display any emotion. On the other hand, cases are reported in which the patient, when aroused from the stuporous state, is jocular and facetious. Euphoria is mentioned by some observers as occurring frequently, especially as an early symptom, while manic-like states with elation are also described. Various authors have mentioned as frequent and striking symptoms, absence of all concern and lack of any anxiety or worry about the illness. In other patients, however, there may be a prominent depressive reaction, fears and even suicidal tendencies. Finally, there seems to occur fairly often spasmodic or uncontrollable emotional reactions, crying or laughing, with or without appropriate affect.

This extremely wide range of emotional reactions indicates that the differences between individual cases are very great or that the mood varies markedly through the different stages of the disease. In giving the results of our observations, the mood changes in the early or acute stages will be considered first and then reference will be made to those which we have found to occur or persist in the later stages; in many instances, the latter might better be regarded as residuals of the disease because otherwise the patient appears to have recovered.

In the early stages, before the onset of the torpor or delirium, mood changes are not often marked. Pronounced depression does not seem to occur although some patients give in retrospect an account of a worried anxious mood during the initial period of the disease. One patient said he felt "tired and sorry and worried" about his condition. Another complained of feeling nervous and vaguely afraid. Climenko has described as a common initial symptom a euphoria with feelings of optimism, well being and unusual desire for work. None of our patients showed in the early stage such a reaction, with the possible exception of the patient in Case 1, who developed with insomnia a busy overactivity and expressed some grandiose ideas about his inventions, but there was no pronounced elation. On the other hand, we have frequently seen euphoria after a lethargic or delirious phase. In Case 5, a stuporous state was succeeded by a euphoric mood, with a great deal of laughter and excessive reaction to jokes and other emotional stimuli. In Case 12, a delirious period passed over into a euphoric condition, a gay happy

mood, with smiling and much laughter which at times seemed almost uncontrollable. Appropriate affect was, however, not lacking and no crying spells occurred. In neither of these cases was there any accompanying general excitement or impairment of judgment, so frequently seen in the euphoric states of paresis and manic-depressive insanity.

Euphoria with added features of a manic reaction does, however, occur. The mood, psychomotor excitement and speech productivity furnish a picture not distinguishable from a manic-depressive excitement. A boy, Case 11, passed through a severe delirium and made a slow and tedious improvement over a period of three months. Then there developed quite a typical mild manic state which has continued for nine months. Significant, however, of the organic foundation for this excitement is the fact that the patient recently, after complaining again of diplopia, had a convulsive seizure. Bremer reported a similar manic-like state in a boy after acute lethargic symptoms of two months' duration. The final outcome in this case was not given. Manic reactions are more apt to appear late than early in the disease and in a sense may be considered as residuals. We have not seen a typical manic state in an adult.

Mood During Stupor.—Of special interest is the emotional state during lethargic and stuporous conditions. Various observers have commented on the fact that with the onset of drowsiness and lethargy, the patient apparently becomes remarkably indifferent and apathetic, and that during the stupor the emotional responsiveness is greatly diminished or even completely suppressed. The patient's immobile face, failure to show any interest and the lack of spontaneity certainly give the impression of an affectless state. Unfortunately, very few observations have been made which include the patients' own account of their feelings during this period. As far as we are aware, no reports have been made on the behavior of the pulse, respiration, pupils or vasomotor system while the patient is subjected to emotional stimuli.

Many patients when aroused will say that they "feel all right" and have nothing to complain about except perhaps that they are sleepy and tired. In many instances, this has impressed us as being a reaction along the line of least resistance, in harmony with the great disinclination to make any exertion, mental or physical. In Case 4, this reaction is typified when the patient says retrospectively, "I was so lazy that I didn't care to have anything on my mind." On the other hand, there are patients who display emotion when specially stimulated, showing that they are capable of a certain response even though they fail to speak.

Tilney and Howe report the case of a girl who in a stupor with mutism and an expressionless face accidentally overheard a remark that

she would not get well. Immediately tears rolled down her cheeks. In our Case 6, the patient for a long time was in an immobile state with masklike face and mutism. When his wife visited him, tears would stream down his face, and often when the physician took his hand, the patient would hang on to it a little, as if he wished him to stay. On recovery, a special effort was made to get the patient to describe his feelings during the stupor. He seems to have felt very dull, was without any initiative and was, so to speak, in a negative, colorless emotional state unless he was especially stimulated in some way. Then he was apt to feel anxious and worried. As he put it, "I was careless like —indifferent—until my wife came—then I would worry about my family." (He recalled that he wept during his wife's visits.) As to his condition at other times he says, "There was really nothing on my mind." If he had to get up or was taken to the toilet he would also become anxious and feel vaguely fearful in reaction to this situation. We can report another interesting observation in this connection. It occurred in the case of a man who, while in a somnolent state in bed at home, was told that his child had been run over by an automobile. He immediately jumped up and ran to the hospital six blocks away where he then had to lie down again because of weakness and somnolence.

It is our view that the most characteristic emotional state in the lethargic or stuporous phase is the apathy which may be extreme. However, just as a patient may be aroused from the sleep so may he be aroused, at least momentarily, by strong emotional stimuli to show an appropriate affective reaction.

Late or Residual Emotional States.—Under this heading we shall discuss what appears to us to be a very significant finding, namely, the persistent emotional alterations which remain after the acute stages of encephalitis have passed, particularly after the delirium and stupor have disappeared—in some instances after convalescence or recovery has apparently been reached.

In looking over our case histories, including the after-history of those discharged from the hospitals, we were surprised to find that only two patients out of eighteen could be considered to have returned to their previous normal condition, with one other case doubtful. The striking feature in all of the fifteen patients that did not recover was a change in the emotional reactions.

Of the fourteen patients who have left the hospital, we find that only two can be said to have returned to their previous normal emotional level (Cases 9 and 15). If we leave out of consideration Case 18, a schizophrenia, and Case 1, a doubtful recovery, we have ten cases

in which there are continuing signs of some definite alteration in character or mood.

In four of these ten, the emotional change is in the direction of a depressive affect, without any lethargic tendencies remaining; in four others the change is in the direction of an emotional elevation or irascibility; in one, there is abnormal stubborness; in another, there is an apathetic reaction, with some persistence of drowsiness.

Moreover, in the four patients still in the hospital (Cases 11, 13, 16 and 17, all of them in state hospitals) we find that the emotional disturbance stands out as a striking feature in the clinical picture, and in two cases, the affective reactions are peculiar and in a sense contradictory. The latter two cases deserve special mention as they may be looked upon as severe psychotic cases and the unusual emotional reactions have been difficult to understand.

In one case, nearly a year after the onset of encephalitis, the patient (Case 13) showed a markedly lazy, sleepy, disinterested state which was interrupted by transient singing and jig-dancing with the admission of feeling happy but without any evidence of special elation in speech or face. The usual torpor and low mental tension were easily overcome by the stimulus of questions and examination and there was then no evidence of impairment of mental ability.

The other patient (Case 17) a year and a quarter after the onset of the encephalitis, was in a depression with suicidal inclinations. The mood, however, was subject to peculiar quick changes which seemed almost contradictory—in the midst of evident anxiety and talk of being killed she could be easily induced to smile, at times even to laugh a little. This peculiar mood reaction suggested at least superficially a lack of correspondence between the affect and the ideas expressed. We do not believe, however, that we have here a dissociation of affect as occurs in dementia praecox. It is rather the quick change of mood, the lability that is misleading, for when the patient talks of being killed she appears actually afraid. The smiling seemed to be, to a considerable extent, beyond her control and was easily elicited by suggestion. She herself said, "I know it is foolish to smile." An underlying genuine affect was shown by her repeated attempts at suicide. There was only slight evidences of organic impairment of the faculties although the patient had a hemiplegia; there was a mild mental tension defect and some variability in memory and thinking capacity.

All in all, our experience tends to indicate that persistent abnormal emotional reactions form one of the most striking features, or after-effects, of the disease. The question has arisen in many cases as to whether or not a permanent damage has occurred in the emotional sphere. Peculiar mood changes are seen in patients who may otherwise be regarded as recovered.

MOTOR PHENOMENA

Many of the motor phenomena of encephalitis have an obvious psychiatric aspect and for that reason need to be considered here. We do not, of course, refer to the various paralyses, tremors, involuntary motions and tic-like reactions, although in the latter an important psychic factor cannot perhaps be excluded. The marked reduction in all voluntary motion, and also to some extent in the reflex activities, gives rise to the picture of the so-called stupor of encephalitis.

Stupor is in no sense a unitary syndrome and, as is well known, a great variety of mental and physical conditions may be involved in a stupor reaction, namely, intense apathy, retardation, blocking, overpowering terror or fear, brain torpor, organic brain disease, etc. From what we have learned regarding the sleep disturbance and the affectivity during the lethargy of encephalitis, we believe that the psychic torpor, inertia and emotional apathy are the most important mental factors in producing the stupor, while the rigidity and certain other muscular symptoms when they occur, are the expression of an organic upper motor neuron phenomenon of the sort seen in paralysis agitans. Typical "cog-wheel" resistance is often present on passive motion.

A great many writers have referred to the appearance of symptoms during the encephalitic stupor which they call "catatonia" or "catalepsy." These terms seem to have been used synonymously without regard for their usual psychiatric meaning. Many have applied these terms, without any further qualification, to describe a stuporous patient in whom it is possible to lift a limb which the patient maintains for a time in the given position. Few if any have mentioned the length of time they have seen positions maintained. Such a symptom must be considered in relation to its setting—the patient's clearness, mental grasp, emotional state, mental content, suggestibility, neurologic status, etc.

The tendency to hold given positions seems to be most often associated with parkinsonian symptoms, especially rigidity. One patient (Case 5), when speaking retrospectively about holding positions, said he felt rigid, that he could bring his arm down but not easily, "it was too stiff."

Tilney and Howe, who have given the most complete reports on the muscular phenomena, identify the flexible hypertonus of the muscles with the parkinsonian syndrome.

Catatonia in psychiatric terminology usually carries with it the idea of negativism, although it is apparently used by some writers on encephalitis to indicate abnormal muscle tonus with a tendency to maintain given positions. We have not observed any symptoms of negativism in encephalitis cases, although it has been shown by Bonhoeffer[7]

and by Kirby[26] that negativistic phenomena and catatonic reactions occur frequently in toxic-infectious disorders and in emotional settings other than dementia praecox. The stupor of encephalitis as we have seen it has, however, none of the characteristics of a typical catatonic stupor such as occurs in dementia praecox. Springlike resistance, release of opposite impulses, fantastic postures, clenched fists, "schnauzkrampf" and cyanosis of extremities have not been encountered in encephalitic stupor. The rigidity of encephalitis does not suddenly disappear and no states of excitement or stereotypies, such as occur in the catatonic forms of dementia praecox, have been reported. Mutism, drooling of saliva, holding of urine, wetting and soiling the bed occur in the stupor of encephalitis and may be mistaken for negativistic reactions. To us, it has appeared more plausible to relate them to the great psychomotor inertia and emotional apathy, a view which is supported by the patients' explanations obtained after emergence from the period of inactivity or stupor.

In retrospect, patients have told us that they did not care to speak because of the effort required and the "lack of voice," and often, in addition, because of a stiffness of the tongue, throat and facial muscles. In retention of urine, it is, of course, necessary to exclude an organic alteration based on a myelitic complication.

The use of the term "catatonia" in describing the motor or muscular phenomena of encephalitis seems to us misleading.

PSYCHOTIC TRENDS AND GENERAL MENTAL CONTENT

Ideas of a specific type are, of course, not to be expected in encephalitis any more than in other organic-toxic-infectious mental disorders. One has only to recall the multiplicity of delusional ideas encountered in syphilitic encephalitis (paresis) and various infectious and toxic psychoses. One pays most attention to the setting in which the ideas occur in these psychoses. We have already discussed the general characteristics of the trends and hallucinatory experiences of the delirium of encephalitis. Transient delusions or fixed ideas that appear independently of the delirium require some mention. Cases in which these occur are the ones in which the possibility of a relation to some other type of psychosis most frequently comes into consideration. In general we may say that definitely formulated and persistent trends are infrequent in the course of an encephalitis.

Vague fears, anxious forebodings and worry about the outcome of the illness are fairly common in the early stage and often appear to express the reactive tendencies of the personality to sickness in general

26. Kirby: The Catatonic Syndrome and Its Relation to Manic-Depressive Insanity, J. Nerv. & Ment. Dis. **40**:694, 1913.

(Cases 4, 5 and 7). On the other hand, the striking indifference or apathy about the situation sometimes encountered seems to be a part of the general lethargic tendency in its mildest or initial manifestation (see under emotional reaction).

Excitements with expression of various trends of ideas may precede the delirium or lethargy, and these may sometimes have many of the characteristics of a manic state. Such reactions are well known in other toxic-infectious psychoses and especially in paresis. In Case 1, there was an acute onset with insomnia, busy activity, productivity of speech and expression of what seemed to be a grandiose trend, talk of inventions of perpetual motion, of a torpedo which could cross the ocean, etc. Later it was learned that the patient had long been interested in inventions and perpetual motion. An interesting feature of this case was the fact that the excitement, which only lasted a few days, occurred mainly at night, and the patient later described a state of mental hyperactivity with his mind running continually on some detail of his work, as he expressed it, "What I was thinking about would stay in my brain all night long." Although there was said to be no clouding of the sensorium at this time, one might think the whole reaction somewhat suggestive of a not fully developed nocturnal delirium with occupational activity.

In Case 15, a circumscribed paranoid trend developed after the stupor, while the patient was in a state of dissatisfaction and worry over her physical condition and the treatment she was receiving. This reaction suggests a schizophrenic episode in a person whose make-up was distinctly of a dementia praecox type.

In Case 18, a very peculiar fantastic trend of religious sexual content followed a typical encephalitic attack (lethargy and delirium). In this case, which we regard as one of dementia praecox, we find distinct evidence of a change in character and a shifting of religious point of view prior to the encephalitis. It is our view that in this case an incipient schizophrenic reaction has been intensified coincidently with the brain disease to a point at which a fully developed psychosis may now be recognized.

In Case 17, a severe and prolonged suicidal depression with paranoid ideas and hallucinations followed the acute phase of the disease. With clear orientation, she accused the physician of wishing to kill her, voices say she was a bad woman, that she "broke the Jewish temple." A very peculiar emotional reaction accompanied the depression. She smiled in the midst of talking about being killed; the patient seemed unable to control this smiling, which could also be brought out on suggestion. She herself characterized it as "foolish." An underlying

strong effect is seen in her repeated suicidal attempts. This peculiar emotional reaction seemed to be quite different in nature from the discrepancy between mood and ideational content so often seen in dementia praecox.

MENTAL GRASP, ORIENTATION AND MEMORY

We have spoken of the mental clouding during the delirium and have also touched upon questions of orientation and mental grasp during somnolent and stuporous states. Observers usually say that when patients can be aroused from sleep or even stupor, they are often remarkably clear as to their environment, etc. The degree of clearness can, however, vary a great deal even within a short space of time. We know that often a patient who appears clear during the day may be delirious at night, in fact, a patient in a lethargic or stuporous state may sink at any time to a lower level of consciousness, mutter and show delirious "dips," for a short or longer period.

Even in patients who are apparently clear when awakened, able to appreciate where they are and to recognize those about them we have found on closer examination there were evidences of interference with the mental processes and difficulty in grasping more complicated things, especially when sustained effort and thinking were required. Facts learned by rote and habitual memories may be at command, while mental operations of a more complex character are quite beyond the patient. This we interpret as a mental tension defect, such as occurs in acute organic reactions generally.

We have given examples of the ease with which a stuporous patient may be stimulated and raised to a higher level of consciousness and emotional activity. In a typical toxic-organic delirium, as for example, delirium tremens, it is, of course, well known that the patient may usually be readily influenced by questions and appropriate stimuli and be lifted to a level where he is able to give quite accurate personal data and other information acquired prior to the onset of the delirium. One of our patients, after the stupor was over and general clearness as to environment existed, took hours to compose a note of a few lines to his wife and made many mistakes in spelling. (Later after full recovery he had no trouble in performing such a task.)

We have found in our cases very little evidence that any severe or lasting impairment of orientation or memory follows the acute stages of the disease. If the brain should be permanently damaged by diffuse or coarse lesions, as is quite conceivable in encephalitis, one would expect some intellectual impairment, or dementia. We have, however, seen very few indications of a deterioration of this nature in any of our patients. We have examined carefully three patients, with severe

symptoms, who remained in state hospitals, for the purpose of determining whether or not a definite intellectual impairment could be demonstrated (Cases 13, 16 and 17).

In one of these (Case 16), there was evidence that a slight mental tension defect remained, but with attention and effort which was easily obtained, the patient showed no impairment in the fields of memory or retention.

In Case 13, the mental tension defect was more marked, with some torpor and disinclination to make mental exertion. He sometimes made mistakes in time relations and in recalling incidental occurrences. However, under the stimulus of questions, he was able to register and retain impressions very satisfactorily and to give accurate information about the remote past—showing that his memory was good.

In Case 17, there was more evidence of an impairment of the intellectual faculties, but it cannot be said to have been of severe grade. There was variability in the accuracy of her memory, and capacity for mental operations. On specific tests, with good attention, the patient could, however, do very well on the memory and retention tests although she complained of a certain feeling of difficulty in mental application.

So far as our observations go, we have not found any marked defects in the intellectual faculties as a result of encephalitis. In only one patient (Case 17) did there seem to be some impairment, this being a patient who suffered a hemiplegic attack during the stupor and subsequently developed a prolonged depression. Whether or not these severe psychotic cases will show later on, more evidences of a permanent organic type of mental impairment is a question which we leave open.

DIFFERENTIAL DIAGNOSIS

In the differential diagnosis, one must take into consideration principally: (1) toxic states with mental symptoms, (2) infections with cerebral complications, and (3) various kinds of organic brain disease with mental disturbance, including disorders of the pituitary gland.

A large array of toxic states and poisonings, lead, carbon monoxid, alcohol and some food poisonings, chiefly botulism, diabetic and uremic states of coma, may require consideration. Various febrile deliria and infectious psychoses may have to be excluded, especially acute cerebral syphilis and meningitis. Numerous organic brain diseases, trauma, tumor, etc., may figure in the differential diagnosis.

The criteria by which these conditions may be separated from encephalitis are the history of the case, the physical signs and the laboratory findings. The psychotic symptoms of encephalitis show nothing in themselves sufficiently characteristic to distinguish them from the mental reactions in other toxic-infectious-organic conditions. In them

all, the acute organic syndromes as outlined earlier in this paper may be present, in each, in various degrees of severity. A single exception to this statement is the reversal of the usual day and night sleep sequence. As far as we are aware this has not been noticed, or at least reported, in conditions other than epidemic encephalitis.

It is, of course, well known that in infectious psychoses and in various toxic-organic mental disorders delirium and restlessness are more apt to occur at night.

SUMMARY

The psychic disturbances of epidemic encephalitis present the general characteristics of an acute organic type of mental reaction, corresponding more specifically to a toxic-infectious psychosis.

In the acute stages of the disease, psychic torpor and delirium are the most frequently observed mental disturbances although other clinical pictures may be encountered, as the Korsakoff syndrome or more complex mental disorders in which various affective and trend reactions give a special cast to the psychotic disturbance.

Two types of sleep disturbance occur, hypersomnia and hyposomnia. The four gradations of the former are drowsiness, lethargy, stupor and coma. Hyposomnia is not common, but is occasionally an onset symptom and sometimes occurs at other stages of the illness. A reversal of the usual day and night sleep cycle occurs, with the result that the patient is insomnic at night and somnolent during the day. It is impossible to draw fine distinctions between degrees of somnolence and states of lowered activity (stupor without sleep). Theories regarding the causation of the disturbances of sleep are inconclusive.

A great majority of patients with encephalitis show delirium at some stage. Transient delirious features during a stupor may be easily overlooked. In encephalitis, the content of the delirium tends to center about habitual trains of thought and occupational activities, but is sometimes determined by somatic sensations. The content of the delirium is usually remembered, while amnesia of different degrees, sometimes "patchy," remains for external events.

Before the onset of lethargy or delirium, mood changes are usually not marked, although certain patients in retrospect have described a worried, anxious mood at this time; and in contrast, one patient at the onset, while insomnic and overactive, expressed grandiose ideas but was not apparently elated. After the passing of the lethargic or delirious phase, euphoria frequently arises and with it sometimes uncontrollable laughter with appropriate mood. Features of a manic reaction are sometimes added to the euphoria and furnish a picture not distinguishable from a manic-depressive excitement.

Depressive reactions in various grades of severity not accompanied by retardation, although in one case with repeated suicidal attempts, have been seen following the stuporous or delirious stage.

In the lethargic and stuporous states, there is apathy and apparent inaffectivity. This is chiefly a disturbance of showing affect rather than an absence of affect. Emotional response of some kind can be obtained from almost every patient, provided the stimulus is adequate. Just as a patient may be aroused from the sleep, so may he usually be aroused by strong emotional stimuli to show an appropriate affective reaction. We have encountered peculiar, and in a sense contradictory, emotional reactions in patients who still have not recovered. Some of these mood reactions suggest, at least superficially, a lack of correspondence between the affect and ideas expressed. We have not found, however, dissociation of affect, such as occurs in schizophrenia. It is rather the quick changes of mood that are apt to mislead; but with these changes, there seems to be always a corresponding change in ideational content.

In all of the unrecovered cases, there were signs of some definite alterations in character or mood. This, in a large proportion of them, constitutes the only evidence of lack of recovery. These continuing alterations in character and mood consist of depressive affects, emotional elevations, irritability, explosive reactions, stubborness, apathy, etc.

The extremely wide range of emotional reactions encountered in epidemic encephalitis probably indicates both that the differences between individual cases are very great and that the mood varies markedly through the different stages of the disease. Our findings are at least suggestive of a lasting damage in the emotional sphere in a considerable number of all cases.

Psychic torpor and emotional apathy appear to be the most important mental factors in producing the stupor, while rigidity and certain other muscular symptoms, when present, seem rather to be the expression of a motor phenomenon of the sort seen in paralysis agitans. The tendency to maintain given positions (catalepsy) is most often, if not always, associated with parkinsonian symptoms. We have not observed any symptoms of negativism in encephalitis cases, although it is well known that negativistic phenomena do occur in toxic-infectious and benign emotional disorders, for example, in manic-depressive psychoses. In none of our cases have we seen a typical catatonic syndrome such as occurs in dementia praecox. The term "catatonia" used by various writers to describe the motor or muscular phenomena of encephalitis appears to us to be misleading.

Ideas of a specific type are not found in encephalitis any more than in other organic-toxic-infectious mental disorders. The multiplicity of

mental symptoms is well known in organic-infectious disorders, for example, paresis, in which a great diversity of clinical pictures arise in association with brain changes of a very definite and specific nature. Definitely formulated and persistent trends are infrequent in epidemic encephalitis, although our series includes two fairly clear schizophrenic reactions, one of which had begun prior to the encephalitis and in the other a latent tendency was indicated in the personality make-up.

In regard to the outcome of mental symptoms of epidemic encephalitis, we have found much evidence of persisting emotional alteration with little evidence of organic mental defects or dementia.

CASE HISTORIES

CASE 1.—*Indication of a psychopathic make-up, "a book worm" interested in perpetual motion schemes. Acute onset, insomnia, headache, mental and physical overactivity; few days of excited talk of inventions and perpetual motion ideas (no delirium). Then short period of drowsiness, followed by feelings of physical insufficiency without emotional depression, oculomotor symptom at onset and some choreiform twitchings. Later peculiar feelings in left hand with some involuntary movements in arms and shoulders. Physical residuals present after nine months, full mental recovery, somewhat questionable. Complains of weak voice and lacks complete insight.*

History.—E. H., aged 27, machinist, single, was admitted to Brooklyn State Hospital, Feb. 29, 1920. His parents were both healthy; one brother stammered. The patient was born in Brooklyn. During infancy he had "marasmus." He was bright at school, finished the grammar grades at 14, was always studious and was called by the family "a book worm." He was such interested in mechanical contrivances, electricity and inventions; he worked some on perpetual motion schemes; cared less for amusement than for his work. It is said, however, that he was not seclusive and that he had many friends. During the war, he served in the Navy and was assigned to duty on a destroyer. Later he was on a submarine. After his discharge from the Navy, he worked steadily at his trade, earning $35 a week. In August, 1919, he had influenza, was given considerable alcohol and is said to have been delirious one night. He recovered without sequelae.

Present Illness.—The last week in February, 1920, he became restless and overactive; could not sleep, would stay up nearly all night going over his drawings and mechanical inventions. He continued to work at his trade until two days before admission to the hospital. He then talked excitedly all of one night about his inventions, torpedoes, steamships and perpetual motion. When put to bed he kicked about and could not sleep. When a physician was called, the patient said he had invented a torpedo which was to go from here to Europe, that he had a scheme by which steamships could cross the ocean by means of electricity generated by perpetual motion. During this time he was oriented and did not seem confused.

He was then taken to the state hospital where he almost immediately became quieter and for four or five days was noticeably drowsy. He could, however, be easily aroused and was able to give satisfactory information about himself, showing considerable appreciation of the fact that he had been upset—"nervous breakdown" as he called it.

Examination.—On the day of admission, he showed some twitchings of the hands and irregular movements of the limbs and head. The temperature was 99.2 F. There was some drooping of both eyelids. The pupils reacted well to light. He complained of double vision and blurring of eyesight. The knee jerks were active; the plantar reflexes were diminished. Lumbar puncture revealed no increase of cells and negative globulin. The Wassermann reaction on the spinal fluid and blood was negative.

Clinical Course.—Five days after his admission, the drowsiness had disappeared, also the diplopia. He said that he felt well again; spoke of having been dizzy, of being unable to read because the letters would get bigger and bigger and then blur. He gave a good account of the onset, with insomnia, restlessness and some headache covering the week before he left home. He described his overactivity at night thus: "After working all day I would start on my blue prints when I got home—I would go right through them until 1 or 2 o'clock in the morning—then some little detail that I would think about would stay on my brain all night long."

Apparently the sensorium was not at any time clouded, and no amnesia for any period of the attack could be established. He did not think that the ideas which he expressed about perpetual motion were unusual or peculiar.

Fifteen days after admission (March 14) he was allowed to go home with his father. The diagnosis was lethargic encephalitis. He did not feel able to resume work at once. He complained of weakness, seemed a trifle dull and lacking in initiative.

Subsequent Course.—April 10, 1920, he was examined. He had not regained his strength and energy. He appreciated this and remarked, "I feel lazy, tired and sleepy—I feel like dropping off sometimes." Also he mentioned that his voice was weak, "My speech just fades away—I just mutter my words." No emotional depression accompanied this subjective weakness.

Nov. 17, 1920, he was again examined. He appeared bright and alert. Recently he had opened a shop of his own. He felt that he had entirely recovered his general strength, but still thought that his voice was not so strong as before his illness. He also complained of a disagreeable drawing sensation localized in the left hand and fingers, especially on the ulnar side of the hand and the outer three fingers. This feeling had developed recently. It was accompanied by occasional involuntary motion of the left arm and elevation of the shoulder. Examination revealed no impairment of muscular power of the hands or fingers and no disturbance in tactile sensibility.

When his psychosis was reviewed he was able to give a good account of what he passed through. He confirmed his previous statements that the onset was with insomnia and busy overactivity and fussing with his drawings and inventions. He asserted that he had not had any hallucinations during the attack. Even at the time of this examination he did not think the ideas expressed during the disturbed period were delusional. He believes perpetual motion is possible and has an idea that it may be feasible to construct an electrically driven ship with small turbines in each side which will transmit a current to a large turbine so that when the ship once gets to going it will be able to generate its own motive power indefinitely.

He recalled the diplopia and drowsiness which he felt for a few days after his restlessness and insomnia subsided.

CASE 2.—*Steady worker. Athletic interests. Cheerful. Oculolethargic type. Benedikt's syndrome. No psychotic symptoms besides moderate stupor*

and early slight transitory delirious features. Short course. Some physical residuals. No mental residuals except possible alteration of character.

Family History.—S. M., aged 18, an auto mechanic, was admitted to Bellevue Hospital, Feb. 7, 1920. His parents, two sisters and one brother are living. There was no history of nervous and mental diseases. He was born in Poland, but came to the United States in early childhood. Here he attended public school, and made ordinary progress. At 15, he went to work. As a worker he was known to be very steady. He had been also an obedient son, good to his parents and free from bad habits. He had been much interested in ball playing and, more than the average for boys of his opportunity, interested in track athletics. In disposition, he was cheerful and not moody.

Present Illness.—His illness began early in February, 1920, with dizziness and diplopia, the latter causing him to attend an eye clinic. He wore a patch over one eye for a few days and then because of sleepiness he had to go to bed. He slept more than was normal, feeling sleepy both night and day. At night, there were occasional brief periods when he was restless and probably somewhat delirious; his family said that he talked "foolish," but were unable to recall what his talk was about. After a week or ten days at home he went to the hospital by street car.

Clinical Course.—In the hospital he presented a typical lethargic state, a condition of semistupor from which he could be readily aroused. He was not negativistic. Neurologically, there had developed a Benedikt's syndrome, a complete ophthalmoplegia externa on the right and a partial left hemiplegia accompanied by hemiataxia on the same side. His temperature was normal and there was a negative serology. The lethargy was of short duration and disappeared after five or six days in the hospital. Though there is record of the "foolish talk" prior to admission, there were no delirious symptoms noted in the ward. His orientation was maintained. He was able to leave the hospital after six weeks.

Subsequent Course.—When seen six months later, it was found that he had no true amnesia for any portion of his illness though he recalled that for a short period things seemed rather "foggy." There was during the illness no feeling of anxiety, no sense of impending danger or fears of death. There was not apparently a real distress over his disabling symptoms.

He returned to work one month after leaving the hospital. He had been slightly inconvenienced by a variable unsteadiness of the left hand (which showed ataxia during the acute stage). He explained that the strength was there but sometimes he could not control it so well as previously. The disturbance was noticed at comparatively infrequent periods.

Mentally, at this time, there appeared to be an almost complete return to normal. He was alert and quick, showed nothing unusual in speech or mood. There was no alteration in his interests or desires for activity, which, as before noticed, were always considerably along athletic lines. If judged alone from interviewing him, there would seem to be almost an unnatural confidence in view of his continuing partial disability. In contrast to this, however, his relatives said that he was different now and much less jolly and goodspirited than prior to the illness. To them he showed a considerable concern regarding his disability.

CASE 3.—*Good make-up. Disease began with diplopia. Great drowsiness Partial confusion—regarding time; clear regarding people. Never hallucinatory.*

Very good recovery after eight weeks. However, residual change in disposition with great social activity and less ambition. Subjective impairment in powers of memory.

Family History.—E. B., aged 23, bookkeeper, was admitted to Bellevue Hospital, March 15, 1920, with no history of nervous or mental diseases.

Previous History.—The patient was a young, industrious, American office worker with good habits. Her education consisted of the common grades in parochial schools, followed by night high school for three years, during which time she learned stenography. Finishing at 16, she went to work immediately. For the last two years she had worked for a firm of brokers, and as an office worker had risen to a place of importance, being in charge of the firm's cash-book and check-book.

Her make-up had in all respects been excellent. Though subject to certain depressed moods, these had never been severe nor followed by any swings toward elation. There had always been a strong interest in social affairs, parties, dancing, etc., also a fair amount of outdoor life. She was a good skater, fond of the surf, though she never taught herself to swim. Her health was always excellent and she had been a self-reliant, energetic type of person.

Present Illness.—Her illness began in February, 1920, with gastric symptoms, followed by persistent dimness of vision, diplopia on reading and much drowsiness. She slept twelve hours each night and a large part of the day time. She knew people quite well but had marked tendency to lose track of the days of the week. Though there was increasing weakness, progressive loss of weight and the great sleepiness, her physician urged that she be compelled to be outdoors a great deal. Walking was forced on her. More than once, she almost collapsed, but for six weeks this policy was persisted in. Finally on the advice of others, she became a patient in the hospital. She went to the hospital alone, making the trip by street car.

Examination.—For the first two days (only), there was a fever of 100 F. The leukocyte count was 7,600; polymorphonuclears, 68 per cent.

Neurologically, there was nystagmus, with slow oscillations to the left, and rapid to the right. There was some impairment in upward movement of the eyes with a tendency to vertical nystagmus. The left pupil was larger than the right; it was also more sluggish to light. There were no changes in the fundi. There was masklike facies. There was a suggestion of a thalamic facial on the left, also some drooping of the right angle of the mouth. There was a twitching of the upper lip—particularly on the left side. There was slight stiffness of the elbow joints. No pathologic alteration of the reflexes of the pyramidal tract signs was noted. There was normal sensory status.

Clinical Course.—There was nothing strange in her attitude or behavior in the ward, beyond a drowsiness which gradually went away after the first few weeks and a slight difficulty with facts of time. She was clear regarding people. During the nights she talked in her sleep, but when awake she expressed no strange or delusional ideas.

By the middle of May, 1920, she had made virtually a complete recovery and was discharged.

Subsequent Course.—In July, 1920, she returned to her usual office work and has been able to carry on duties presumably satisfactorily to her employers. But when interviewed in October, 1920, she complained that her "memory is very bad and not improving. I am more absent-minded than anything else." She explained the fact that she had done the same variety of work without

complaints from employers by saying, "Yes, but I make notes of everything and do not trust my memory."

Tests revealed no demonstrable defects. Memory for the remote past was strikingly good when questioned on family and personal dates (poor when dealing with school knowledge).

She repeated seven digits with rapidity and persistent accuracy, and showed no attention defect.

Regarding mood, one noticed a tendency toward elation. She remarked with considerable gusto, "I am more optimistic than I was before and I'm happier." That this mood was not obviously pathologic was shown by the fact that the sister had not noticed it. Yet when questioned, the sister admitted that the patient was going more to theaters and to dances than before her illness, that she needed no urging to join any social occasion and that she was keeping poor hours. The patient then explained, "But, I feel so restless. I want to be on the go all the time." This restlessness showed itself also in an increased attention to her clothes and to the dressing of her hair, etc.

The mother of the patient, who was also interviewed, believed that the patient since her illness had shown a poor memory. But such examples as she gave were better examples of a certain carelessness, hand in hand with her mood. The mother complained that the patient "hasn't the right ambition yet" —as she did have prior to her illness.

The patient recounted all features of her illness, and showed absolutely no amnesia. She was able to recall the dreams which caused her to talk in her sleep so persistently. "I imagined terrible things at night, but never expressed them—that accidents might happen to the family. I could see them happening."

CASE 4.—*Meager education. Sociable disposition. Onset with nervousness and vague fears, then somnolence. Oculomotor symptoms, partial left hemiplegia and pseudoparkinsonian syndrome. No delirium or delusions. Short course. Following illness has shown a change in mood and lack of interests with seclusive tendencies. Slight physical residuals.*

Family History.—R. S., aged 42, Hebrew, shoemaker, admitted to Bellevue Hospital, Sept. 29, 1919. The family history was negative for nervous and mental disease. He was born in Russia. His schooling was scanty and included barely two years of study. Prior to coming to the United States (at the age of 27) he had spent six years in South Africa. His work there was divided between harness-making and cobbling. For a time, he owned a small shoestore of his own. His habits were quiet. He disliked drinking, took small interest "in women" and married at 28. As a boy he is said to have had good health and to have been active. Yet he avoided rough games and found his chief pleasure in rather excessive reading. He could skate and swim. Prior to his illness, he took a lively interest in socal affairs, often played card games, etc., with a neighborhood circle. He was always known to be jolly and not given to gloomy periods of any character. In later years, he lost interest in religious affairs though previously he was taught and was inclined to be orthodox.

Present Illness.—His illness began one month before admission to the hospital with severe headache, "like neuralgia," and occasional vomiting. He was nervous and "felt afraid" in a vague way without clear-cut ideas regarding any cause for this. Although he was sleepy, he did not stay in bed. His vision was dimmed and off and on he saw double. He came to the hospital by automobile.

Examination.—On admission he was more or less somnolent with slow reaction time, although able to answer questions rationally. He showed a bilateral ptosis with inability to converge or depress the eyeballs to a normal extent. There was a slight deviation of the tongue to the left, weakness of the left side of the face, with a bilateral masklike expression and weakness of the left arm and leg. However, there were no pathologic reflexes. The sensory findings were normal, likewise the fundi. There was fever for only a few days, not rising above 101 F. The serologic examination showed 7 cells per cubic millimeter in the spinal fluid.

Clinical Course.—There was no disorientation; also no hallucinations or delusional ideas. There was continued insight. During his stay in the hospital, coarse tremors developed in the arms and hand, and when he was allowed to be up, his posture and gait showed the characteristics of paralysis agitans. The course of his illness was brief, and after six weeks he was discharged as cured.

Subsequent Course.—A year following his recovery, the patient was again seen. He was without physical residuals except for a certain slight rigidity of the upper extremities and a tendency for the hands to take the parkinsonian position. Mental examination revealed that he talked freely, had good retention and memory, entertained no abnormal ideas, in fact he made quite a natural impression. However, he said that he was now considerably more quiet than before his illness. His sister and all of his friends mention this to him and continually urge him to liven up and take more interest in social affairs. This he thought he was doing. He did not feel depressed nor worried. He took his usual interest in daily papers.

Although he got back to work one month after leaving the hospital, he found during the ensuing several months that he was unable to mix with people. He said, "During the winter I used to hide from people. I was always looking for a chance to be alone." Although shy regarding social affairs, he was not ambitionless nor indolent. There was no lessened ability to work parallel to the lessened ability and lessened desire to mix with people. Although he could think of nothing definite to cause him concern and even denied feeling sad, when asked why he was more quiet than before he said, "My mind brings more worries than it did before." No better explanation of his loss of liveliness could be drawn from him.

For the period of his illness there was no amnesia. The vague fears which he felt early in the attack did not relate to ideas of dying. Such a thought did not enter his mind. In fact, he was not at all distressed at being sick. Touching on his mental state during the course of his illness he said, "I was so lazy that I didn't want to have nothing on my mind."

CASE 5.—*Even-tempered. Optimistic. Regular habits. Efficient. Slow onset oculolethargic syndrome; later very severe pseudoparkinsonian syndrome. General rigidity. Kept given positions. Unresponsive. Stupor. Later euphoria with uncontrollable laughter. In retrospect, depressed mood during lethargic and stuporous stage. Though physical residuals (pseudoparkinsonian) are incapacitating for work, there are no demonstrable mental residuals except a possible abnormal stubbornness.*

History.—H. S., aged 32, violinist, admitted to Bellevue Hospital, Oct. 15, 1919, with a negative family history, was born in Russia, received only a poor schooling, in all about four classes, but learned to play the violin by which means he made his living. He came to the United States at the age of 23, and

over here made a fair living because he was leader of his own small group of musicians and had plenty of work to do, playing for dances, etc., on the lower East Side of New York where he lived. He was married and there were two healthy children.

He had always been steady and regular in habits, never drank, used cigarets in moderate excess, and had never had any venereal disease.

In make-up, he was even-tempered and good natured, good to his family, optimistic, never gloomy nor worried. He was not easily angered. He read the newspapers daily because he liked to keep informed. He inclined to attend the synagogue regularly, even sometimes twice a week. He was fond of social affairs, other than card games; always felt well and was not a complaining type. In 1918, he had influenza for ten days without any complications or residuals.

Present Illness.—The patient's illness began in September, 1919, with severe headache, weakness and drowsiness. His head felt as if there were wheels in it. There was no diplopia at that time and no ocular symptoms later. For three weeks he was in bed at home. He knew his wife and others, was not confused regarding time and did not express any peculiar ideas. He was able to walk to and from the lavatory until the last few days prior to admission to the hospital. It had also by that time become difficult for him to take food and almost impossible for him to talk owing to the rigidity of the tongue, lips and face. Similar rigidity was general throughout the body musculature on admission.

Examination.—In the hospital, the fever of 101 F. remained for only one day. The leukocyte count was 12,200, 69 per cent. polymorphonuclears. The blood pressure was 120; the Widal test was negative. Neurologic examination revealed no defects in the extrinsic ocular movements. The pupils were normal; the fundi negative; the facies masklike. There was an unwinking, reptilian stare. Marked muscular rigidity was noted in all extremities, and in the facial muscles, also in the tongue. Lateral movements of the lower jaw were impossible. There was a tendency to maintain the arms in given positions. There was increase in the ligamentous tone; there was no stiffness of the neck. The Kernig sign was absent. Deep reflexes were obtainable when muscular relaxation was secured. Plantar reflexes were normal. No demonstrable sensory changes were noted. The blood Wassermann reaction was negative. The spinal fluid showed 20 cells, a negative Wassermann reaction and the colloidal gold curve was 1122100000.

Clinical Course.—Four days later there was noted a midbrain tremor, affecting especially the left upper extremity, also a thalamic facial paralysis on the left.

After thirteen days in the hospital, the parkinsonian tremor was present in all extremities and still more marked in the left upper extremity. During approximately the first ten days there was marked mental torpor and drowsiness. This diminished. The facial expression brightened, although the thalamic weakness persisted. At this time the mood changed and the patient became euphoric. With this, there was uncontrollable laughter. Other patients noticed him laughing impulsively for, as they said, "two hours at a time." He felt happy at this time and the laughing, although he could not stop it at will, was in accord with his feelings. Joking about it among the patients and things in general in the ward are believed to account for the length of time it frequently lasted.

During the period in the hospital when the patient was stuporous he did not seem to understand questions, but in retrospect he says that he understood everything and that he was unable to talk because of the rigidity of his tongue, lips and all his vocal apparatus. The patient was not delirious, did not react to delusions or hallucinations, and showed no negativism.

When questioned during convalescence, the patient was able to give a good account of his trip to the hospital and later happenings. He said he "felt tired and sorry" and "worried about" his illness.

Regarding the maintenance of an imparted position he remembered that physicians put his arm in the air, and he kept it there because it was stiff. Although it was possible to bring it down, it was not so easy to do so. He "was glad to bring it down but couldn't easily—it was too stiff."

Subsequent Course.—When seen a year later (October, 1920) he showed a severe residual parkinsonian syndrome with the right hand and arm in the position assumed in paralysis agitans, variable fine tremors, masklike facies and an annoying tendency to fall forward in walking. These features had entirely precluded his return to violin playing and had kept him from work.

Mentally at that time, his attitude was normal, except for a possible stubbornness in refusing all work except violin playing. He had a good interest in affairs and quite excellent memory. He read newspapers each day and gave points concerning several matters quite accurately. He was able to repeat six digits rapidly. His difficulty in performing small tasks in arithmetic was considered in keeping with his training. He was not emotional when telling of his condition and was not unduly anxious.

CASE 6.—*Good make-up with slight indication of emotional instability. Lethargy, general rigidity and unilateral limb pains. Transitory slight confusion and delirium. Next followed prolonged immobile state, masklike face, mutism and gazing. No catalepsy or negativism; tearful on stimulation. Vague fears and anxious ideas, few hallucinations. Recovery with little amnesia. Possibly more indications of emotional instability than before illness.*

Family History.—T. R., aged 31, freight handler, admitted to Manhattan State Hospital, April 26, 1920, with a family history that was negative for nervous and mental diseases, was born in Ireland. He was healthy as a child, received instruction in the common school as far as the fifth reader and was an average pupil. He did laboring work, was industrious and steady. He came to the United States seven years ago and has since worked as a freight and express handler. In disposition, he was said to be even-tempered, friendly and sociable. He was regarded as rather "tender hearted" and easily moved to show affection. He drank whisky moderately. He was married three years ago and had two children, living and well.

In the spring of 1918, he had influenza, was in bed two weeks and recovered without sequelae. He never had any nervous or mental trouble prior to his present sickness.

Present Illness.—Early in February, 1920, he was taken sick with fever, pains in the head, neck, right shoulder and arm. He felt drowsy and "dopey" and was in bed off and on until his wife gave birth to a baby, February 22. He then made a great effort to keep up and continue at work. About the middle of March, however, he gave up and went to bed. He then felt very sleepy and heavy, and continued to have pains in his right shoulder, arm and leg. He was weak and shaky. For two or three weeks, he lay in bed almost immobile; he rarely spoke and had a peculiar blank facial expression. Occa-

sionally, he would express some vague fears and anxious ideas. He said someone was after him; he thought people in the hall talked about him but he could not understand what they said; he imagined he was to be taken away. A few days before he was sent to the hospital, he was more apprehensive and uneasy, talked of being killed by some one but did not specify by whom. He was taken to the psychopathic ward of Bellevue Hospital. He there appeared very weak, perspired profusely, and most of the time lay very quietly in bed in a rigid attitude gazing at the ceiling. He did not answer questions, although he appeared to make some effort at speech by mumbling, and he would understand and obey simple commands such as to show the tongue. There was some general stiffness when passive motion was attempted but no catalepsy.

Examination.—April 26, 1920, the patient was transferred to the Manhattan State Hospital. He then had a fever of 102 F. which gradually declined to normal in five days. The pupils reacted to light and the eye movements were free. The knee jerks were equally increased. Spinal puncture gave positive globulin, no cells and a negative Wassermann reaction.

Clinical Course.—For four months following his admission to the state hospital, the patient continued to exhibit a marked reduction in activity, but without muscular stiffness, resistance or catalepsy. He never drooled saliva, held his urine or showed other negativistic behavior. He rarely changed his posture in bed, requiring to be spoon-fed and for a time urinated and defecated without making any effort to go to the toilet or to let his wants be known. He did not appear to be drowsy during the day, as he kept his eyes open most of the time and usually gazed in one direction. The masklike facial expression was striking. When approached he would, however, give some attention, as shown by the eye movements, and on various occasions he displayed considerable emotion even though he would not speak. For instance, during his wife's visits, tears would roll down his cheeks and often he would hold on to the physician's hand and act as if he wanted him to remain. Sometimes he would mumble as if he were trying to say something. On one occasion during the early part of his hospital residence, he answered a few questions after much urging. He seemed then to be rather perplexed and unclear as to the situation. He was not certain whether he was in a hospital or not, said it was 1916 or 1920, could not tell how he came to the hospital or how long he had been here. He spoke of hearing Jewish and Italian people talking, also of hearing his wife's voice. Once in a confused way he spoke of his citizenship papers being on an empty chair by the bed. (He had been naturalized just before his illness. After recovery he said he imagined if he were known to be a citizen he would be better treated.)

During August, 1920, the patient began slowly and gradually to emerge from his inactive state. Nov. 20, 1920, he had apparently recovered and was ready to be discharged.

Subsequent Course.—He was bright and alert and capable of doing considerable work without fatigue. He had excellent insight and repeated tests failed to show any impairment of his general memory, retention, attention or mental capacity. His interests were keen, he was anxious to return to his family and his emotional reactions were adequate and stable with perhaps one exception: when talking of his wife and children, the separation from whom he felt very much, his eyes were apt to fill with tears. He said it was his nature to be affected easily and this was confirmed by his wife (see personal history).

On reviewing his illness with him it did not appear that there was any definite period of amnesia which could be defined. He recalled very well all of his movements and most of his symptoms.

He mentioned among the early symptoms a heavy, sleepy feeling and pains and stiffness, especially on the right side of the head and body (shoulder, arm and leg). Later he felt vaguely afraid that something would happen to him, that he would be taken away, perhaps killed. Hallucinations were infrequent, occasionally he heard remarks which he thought referred to him. Once he imagined he heard his brother speak, saying, "Oh, Terry, I dread the operation table." On another occasion he thought he saw his wife outside walking across the lawn (illusion?). He recalled that he lay still and inactive, gazing and not responding to questions. His explanation for this conduct was that he felt extremely weak and did not care to move or exert himself. He also said that he was vaguely fearful of attendants and other patients, thought he was disliked and thought it was best to keep quiet. He thought he realized where he was fairly soon after he came to the hospital, but he could not keep track of time. He could not give any reason for his failure to speak except that he was timid and weak, as he put it, "so sick and deadlike that I didn't care for speaking." As to his mental content during the stupor (aside from the occasional fears), he said, "There was really nothing on my mind at that time." He seemed to have felt very dull and without initiative, he was in a negative, colorless, emotional state unless he was stimulated in some way, then he was apt to feel anxious or worried. As he put it, "I was careless-like (indifferent) until my wife or my friend came—then I would worry about my family." (He was often tearful during visits.) In a somewhat similar way, if he had to get up or was taken to the toilet, he would become anxious and feel vaguely fearful under this situation. As he improved he worried a great deal about his family and his circumstances.

CASE 7.—*Good make-up. Efficient. Sociable. Moderately alcoholic. Tendency to worry. Myelo-encephalitis, cervical localization with lower motor neuron signs. Also oculomotor symptoms and pseudoparkinsonian development. At onset, delirious episode. Lethargic course over eight weeks followed by depression, the latter persisting after five months in moderate nonincapacitating form with fluctuations in intensity.*

Family History.—J. B., aged 35, a structural iron-worker, was admitted to Bellevue Hospital, Feb. 23, 1920, with very little information. There was no known mental or nervous disease. The patient was born in Denmark and had had only a few years of schooling. Having been made an orphan, he went to work at the age of 10 years variously as a farmhand and woodsman. Later, after emigrating to the United States, he learned the structural iron-worker's trade and in this made a good living.

Twenty years ago he had typhoid fever and subsequently gonorrhea. Syphilis was denied by name and symptom. He was married and there had been one healthy child. Though he drank considerably, he asserted that he could count on one hand the times he had been intoxicated. He was inclined to rather excessive venery, yet ceased all promiscuity on marriage. His physical strength was above the average and he described himself as "a hearty eater and a heavy worker."

He always had the reputation of being a jolly fellow—one inclined to banter on festive occasions, but he was a hard and steady worker. As a child, he was taught religious ideas scrupulously (Lutheran), and though he never at any time acquired the habit of church attendance, he always retained a religious

feeling about things so that he still each night said the prayers which he learned in childhood. Though he denied that he was ever subject to depressed periods, he told how he was always more inclined to worry about things than his wife. For example, when he was in good health and had no reason to fear unemployment, he would always feel uneasy after finishing one job until he secured another. As regards principles and character, he was a man of more than ordinary steadiness and reliability, largely owing to happiness of his domestic life. He led a tranquil, contented existence in spite of his hazardous occupation.

Present Illness.—Early in February, 1920, he began to complain of pains in his arms and shoulders, the pain being more severe on movement and pressure. After this had been present for about a week, he was compelled to quit work. At this time there was a beginning weakness of the arms. There was diplopia, then for a time some delusional ideas (see below).

Examination.—On Feb. 23, 1920, he entered Bellevue Hospital and showed the following neurologic status: The pupils were small and irregular, but reacted to light and were in accommodation. The fundi were normal. Diplopia had developed ten days before. Eye movement was well carried out; there were nystagmoid twitchings in the external lateral position. There was a suggestion of right facial weakness, tremor of the eyelids and tongue. The face was flushed and masklike. There was great weakness in both arms. The extensors were more affected than the flexors. There was weakness of the serratus magnus, greater on the right. The right scapula was winged. Triceps reflex was not obtained; biceps reflex was exaggerated, the left more than the right. The supinator reflex was normal. There were fibrillary twitchings in the arms. The trunk was free from signs. The abdominal and cremasteric reflexes were sluggish. Knee reflexes were very active; ankle reflexes were present. There was plantar flexion on each side. In sensation tests he cooperated poorly. Nothing was made out except a possible hypesthesia to pain on the outer surface of arm.

The white blood cells were 10,400, 60 per cent. polymorphonuclears.

The spinal fluid showed 40 cells, lymphocytes, with a negative Wassermann reaction and a positive colloidal gold curve 0001210000. The incidence of the infection at the cervical enlargement was considered due possibly to his old infection (typhoid) in part, and in part to his occupation. Pseudoparkinsonian features were superimposed as well.

His temperature in the hospital was scarcely above normal—only 100 F., for a few days. While in the acute stage, he showed a somewhat lethargic state. There was no delirium in the hospital nor delusional ideas. His somber quietness in behavior matched his expressionless face.

Subsequent Course.—In retrospect we find that during his acute illness he was very anxious about himself and thought that he was going to die. His feeling was one of great depression and he was at no time euphoric.

In September, 1920, and later, it was found that residuals of the pseudoparkinsonian picture remained along with persistent great weakness and atrophy of the shoulder girdle. Because of the latter, chiefly, he had been unable to return to his well-paid work as a structural iron-worker. Instead, he was making a meager living, barely able to keep his wife and child in actual necessities, by working as a menial helper in a billiard room. In view of the great ill luck which this amounts to, he had made a fairly good adjustment. Though worried and gloomy he was not despondent or without hope. With

considerable good spirits he anticipated an ultimate complete recovery. When he was interviewed he said, "I haven't smiled for six months. Since I was in the hospital I can't be jolly—it is something I'd like to be but I can't." Later, "A joke don't seem funny to me. I see the idea all right but it don't seem funny." This was, as far as observed, never accompanied by retardation or feeling of hopelessness so characteristic of a manic-depressive depression. This sort of depression is actual and pathologic even though warranted by the economic facts already mentioned.

In this case at the onset, there were some peculiar ideas of a delirious nature for a few days. He imagined that he had three arms and that his arms were bleeding. One night, suddenly, he called to his wife, "Wipe the blood off my arm—that other arm" (meaning a third one). (It seems likely that the cervical lesion and probable coincident cervical root involvement determined the character of this delirious experience.) In retrospect he had full insight into it.

One could discover no impairment of retention or memory defect. He had a partial amnesia for the first three weeks of his illness.

CASE 8.—*Make-up: quiet disposition and mild degree of intellectual subnormality. Onset with diplopia and vision difficulty, then nocturnal restlessness, insomnia and mild occupational delirium; in day time drowsy but mentally clear. Rapid improvement within few days, but after eight months showed persistence of tendency to somnolence.*

History.—T. D., aged 16, press-boy and truck driver, recently a sailor in the Merchant Marine, was admitted to the Brooklyn State Hospital. His father was a chronic alcoholic; his mother was also intemperate and drifted away from her family. He was born in Brooklyn, attended school irregularly and was for a time in a children's home. At the age of 15 he had only reached 7 B grade in the grammar school, indicating that he was retarded. Backwardness at school was confirmed by the patient who said that he was very slow at learning. Later at work, he was, however, quite capable, earning as high as $30 or $40 a week as press-boy.

In 1919, he made a trip to France as a sailor on a steamship, returning in November. Since then he had been working as truck driver.

He was noticeably quiet in disposition, but exhibited no other peculiarities of temperament. He was strong and robust, never had any serious physical illness, and had had no previous nervous or mental trouble.

Present Illness.—The onset was rapid, occurring the latter part of March, 1920, three weeks before he was admitted to the state hospital. He first developed vague symptoms of malaise and sore throat. He complained of his eyes (diplopia) and was taken to an oculist. He then stayed home from work and began to act in a peculiar way. He sat in the house all of one day playing an harmonica and falling asleep every few minutes; every now and then he would drop his instrument as he dozed off. He kept this up so long that he rubbed the skin from his lips. After this for a week he was restless and talkative by night, but as a rule quiet and drowsy throughout the day. He had fever which reached 101 F. During the night, he would run about the house singing, whistling and talking. He spoke a great deal of his trip on the sea, wanted a girl, frequently washed his hands and brushed his teeth. The doors had to be locked in order to keep him in. An occupation delirium was quite clearly indicated. Once he got out on the roof, thought he was on a ship, tried to climb the chimney and said he was putting up a rigging. He imagined he had very large hands. Once he lit matches under the bed

looking for money which was not there. During the day he gave little trouble, would stay in bed or doze in a chair, and although somnolent he could be aroused, and he then appeared quite clear mentally. The patient after convalescence said of this period, "I was out of my head at night but sensible in the day time."

About a week after he became disturbed, he was taken to the Kings County Hospital, Psychopathic Ward, March 31, 1920. There he was in a variable state of clearness, at times spoke of being on a ship, again realized he was in a hospital and that something was wrong with him. When asked how he felt, he said, "I feel dazed in the eyes—I went to a doctor and he put something in my eyes—next morning I saw double and it's been that way ever since."

Some of his replies indicated that he was either facetious or that he would fabricate on suggestion. When asked about going up on the roof he said, "Yes, that's all a fairy tale—I went on the roof to put the screen over the glass—the doctor tells us to carry up the screens and we do it." When asked if he was on a boat last night he said, "Yes, we were out on a submarine chaser."

Physical Examination.—This was made in the observation ward and revealed nothing of importance except slight external strabismus of the left eye.

Clinical Course.—From the observation ward he was sent to the Brooklyn State Hospital, Feb. 11, 1920. By this time he was improving. The nocturnal restlessness and excitement had disappeared. During the day he was drowsy and inactive when left to himself. When talked to he was attentive and responsive. He was clearly oriented, his memory for remote events was good, personal data were given correctly, and he was able to recall a great deal of what had happened during the time that he was mentally disturbed. In fact, no distinct amnesic period was established. He said that he had felt dazed at home, imagined that he had been on a ship, that his money was under the bed, etc. He recalled no auditory hallucinations, although some visual hallucinations and illusions had occurred during the acute psychotic period.

The physical examination was essentially negative. Strabismus and diplopia had disappeared. The deep and superficial reflexes were normal.

His improvement continued and March 7, 1920, twenty-five days after admission, he was discharged much improved, there still being apparently some slight dulness and sluggishness.

Subsequent Course.—When examined, Nov. 17, 1920, he had no physical residuals. The eye movements were normal. For a time after leaving the hospital, he worked as a grocery clerk, but he gave this up and at the time of examination he complained of a general weakness and said that he felt sleepy during the day. He was not working; showed no depression but was somewhat apathetic. He was mentally clear and able to give a good retrospective account of his illness. He recalled very well most of his behavior, even during the period when he appeared somewhat delirious. He had good insight; could not be said to have returned entirely to his previous condition on account of the persisting feeling of weakness and greater desire for sleep than formerly.

Dec. 8, 1920, he had returned to work and did it well although there was a tendency to fall asleep when he had an opportunity. He also complained of occasional blurring and dimness of vision.

CASE 9.—*Good make-up, efficient at work. Sudden onset with dizziness, difficulty in vision and equilibrium, inability to gage distances. Could not sleep*

and developed in a few days an excitement, heard God's voice and talked on religion. Then a violent delirious phase of short duration followed by a few days of drowsiness. Recovered in two weeks with amnesia for delirious phase. Continued well after eight months.

History.—J. R., aged 24, colored, gas-meter indexer, admitted to the Brooklyn State Hospital, April 16, 1920, with unknown family history, since he was left an orphan when an infant, was born in New York. Early development was uneventful. He went through the grammar grades and had two years at high school. He was considered bright and intelligent. He had a lively disposition and good habits, earned $20 a week at his work. There had been no previous nervous or mental trouble.

Present Illness.—The onset was abrupt on April 4, 1920, when he complained of a peculiar dizzy feeling in the head and had immediately some difficulty in vision, stumbled and could not gage distances. He had much trouble in going up and down stairs. He felt as if objects about him were moving. He had to hold on to things for support and it was difficult for him to keep his balance. During the next few days, he complained of noises in his head and imagined he heard music, also he thought God spoke to him. He was restless, could not sleep, talked on religious subjects and said he was saved. He seemed to be confused and at times fearful; he thought something would fall on his head. He was not drowsy. He had some fever at this time. Finally he became so disturbed that on April 9, 1920, he was taken to the Kings County Psychopathic Ward.

He was then excited, noisy and violent, talked in a confused manner, at times laughed, seemed absorbed with hallucinations and paid little attention to questions. He would make such remarks as "These are my things here—my engines and stuff and a lot of goods." This excitement was followed in a few days by a drowsy condition, and April 16, 1920, he was transferred to the state hospital.

Examination.—He then showed a somnolent tendency but could be aroused easily to answer questions. He was approximately correct as to time and knew where he was, but could not tell how long he had been in the hospital (said three months when it was less than a day). He seemed to appreciate that he was sick and spoke of going out of his head. He said people tried to get blood out of his system and thought that upset him. He spoke of hearing God's voice.

Physical examination showed: temperature, 100 F.; active knee jerks; normal eye movements; pupils reacted promptly. Lumbar puncture revealed 7 cells per cubic millimeter and positive globulin. Wassermann reaction was negative on spinal fluid and blood.

Clinical Course.—He remained in a drowsy state for a few days then brightened up, became clearly oriented and had no further hallucinations.

On April 24, it was noted that he appeared to have almost recovered. There was a tendency to sleep during the day and a feeling of lightness in the head. He was able to give a good account of his previous history and present illness. He recalled getting sick on Easter Sunday in church, of being dizzy and hardly able to keep his balance. He was amnesic for the few days spent in the observation ward and did not recall his transfer to the state hospital. He could give little information about his delirious ideas or hallucinations, but recalled that his mind ran on religion and that he imagined God spoke to him.

May 2, 1920, he left the hospital fully recovered with no physical or mental residuals.

Subsequent Course.—Dec. 5, 1920, he was examined and found to be in good health both mentally and physically. He had resumed his work and was just as efficient as before his illness. There was no indication of any change of mood or disposition.

CASE 10.—*Eleven year old boy of previous timid, effeminate make-up. Oculolethargic syndrome. Slight thalamic facial weakness. At the onset, insomnia, and, for three days only, confusion with fears and excessive anxiety. Drowsiness. Subsequent to recovery noticeable increase in pugnacity and aggressiveness. School work less satisfactory for several months, probably due to lack of application. Return of former interest.*

History.—T. M., aged 11, schoolboy, admitted to Bellevue Hospital, Feb. 9, 1920, was one of three children. His parents were living. In the father and father's brothers there was a history of persistent excess in alcohol as long as it was available. The mother's family showed much superior stability.

Previous History.—This was negative except for poliomyelitis at 2½ years with complete recovery. He also had enuresis, which was still present, although in recent years less severe than formerly.

The mother complained that he never got along well with the boys, that he stayed to himself and never learned how to take his share in their sports. He had never learned to fight and the mother believed that she was "to blame for making a coward of him" because she overimpressed on his mind the idea that it was Christian-like to suffer in silence. His habit was to spend every odd minute of the time reading. When school was out he would go back home and read and his mother often wished he would stay outdoors in the air, and exercise. He occasionally did play ball with the boys in the park.

Present Illness.—In February, 1920, he developed diplopia and headache, became restless and had difficulty in sleeping. He began behaving strangely and became disoriented. He imagined that changes had been made in their house; he would wander about the house and appear surprised to find things as they were. His ideas soon related themselves to religious trends and he imagined that dire things were coming to the church. With his beads in his hands he would kneel before "stations," which were imaginary, and pray. To his mother he said, "They are going to chop down the church." At this stage he began having marked dimming of vision. How complete that was was not stated but it appears to have controlled him when he said, "I can hear my mamma, but I can't see her." He found a fur coat hanging in a closet and said that it was his mother, although he could not see her face. Other ideas related to his school affairs and he kept saying that the boys had stolen his books. The mother could not say how clear the patient kept regarding time, dates, etc., during that time.

The period of such symptoms was brief—only three days and nights. They were, in this instance, distinctly phenomena of onset.

On February 9, he was brought to Bellevue Hospital. He went to sleep immediately on admission and was aroused with much difficulty. He ran an uneventful course, never appearing extremely ill, although he was kept in bed for four weeks. His symptoms were the oculolethargic combination with slight left ptosis, diplopia, no (demonstrable) ocular limitations, slight left facial weakness, more apparent on motion, and moderate tremors in the hands

—left more than the right. Although on admission his temperature was 103-104 F., it became normal after two or three days. The laboratory findings included a spinal fluid cell count of 22 and a zero colloidal gold curve. For two weeks, the patient showed drowsiness in the hospital. There was difficulty in arousing him, but he always knew his mother when she visited him. He did no more talking regarding the ideas which have been mentioned and he at no time in the hospital displayed delirious symptoms. There was no so-called catatonia in the hospital. March 18, 1920, he was discharged from the hospital.

Subsequent Course.—When interviewed in September (six months after discharge from hospital), it was found that the patient was different in the following particulars since the illness: The outstanding difference was regarding his timidity. Instead of avoiding scraps, in his mother's opinion, he was seeking them and was taking pleasure in this new aggressiveness. The latter also might be the source of a new sort of impertinence on his part. He was no longer docile, was "bold and sassy" to both his parents. He had grown argumentative and with this had developed an excessive curiosity regarding the neighbors and even a tendency to believe they were encroaching on the rights of his own family. When this was talked over it appeared to amount to nothing more than an overpugnacious reaction to small real conflicts of interest. On the other hand, the patient was slower about doing things than previous to his illness, and according to the teacher did not learn in school so readily. This was thought to be due to lack of application. The patient himself said that he could do as well as before. During the summer he worked for a few weeks successfully in a grocery store.

In talking to the patient himself at this time, it was impossible to discover anything abnormal as regards mood, interests, ideas, etc.

Retrospectively, the patient could not recall the ideas which he had at the onset of his illness. He did recall the period of his stay in the hospital and his trip to the hospital in an automobile. He recalled his drowsiness.

In December, 1920, the patient was seen again. He was doing better at school and had more interest. He was still pugnacious with his companions and more cantankerous at home than before his illness. He was more self-assertive.

CASE 11.—*Bright and well behaved boy of 9. Sudden onset with headache, vomiting and diplopia, followed by an hallucinatory delirium. This was interrupted by a somnolent period of a week, then he became again actively delirious for a few days. On clearing up he was weak and irritable for three months during which time it was noted that he had strabismus of the right eye and involuntary movements, and a parkinsonian gait. Then developed a marked hyperkinetic state with elation, distractibility, pugnacity and overproductivity of speech. Remarkably keen and alert. Orientation, memory and mental ability show no impairment. Excitement continues after lapse of a year.*

History.—A. G., aged 9, schoolboy, admitted to the Manhattan State Hospital, Sept. 24, 1920, with no history of nervous or mental disease for two generations, was born in New York City, the second of four children. He was healthy as a baby, walked and began to talk at 11 months. He had whooping cough at 3 years. He perhaps cried more than the other children, but as he developed he was regarded as a good boy, easily managed, not irritable or cranky. He was fond of play with other children, was well liked by everybody.

When 7 years old, he entered school. He liked to go, learned quickly and never missed a day. His teacher made no complaints about him. He was promoted each time the class advanced.

He had had no previous sickness of a nervous or mental nature.

Present Illness.—The sickness developed abruptly about Christmas, 1919, and was ushered in by fever, headache, vomiting, diplopia, and on the second day delirium. There was "weakness in both legs"; when up, he staggered "like a drunken man." The right eye was turned. He became quieter in a few days and for about a week slept day and night. After this he was again restless and "talked out of his head." He did not seem to recognize his people, at times he swore, again he prayed, at night he spoke of seeing devils and angels. He soon quieted down, but was very weak and irritable. He slowly gained strength and at the end of three months was able to be out of bed.

He was then entirely different from his previous normal state. He exhibited an uncontrollable temper, was very active and quarrelsome. He was extremely restless, on the go continually during the day and slept little at night. He ran about the house and was constantly getting into mischief. If taken out for a walk he ran about the street, would jump on people (strangers) and kiss them; he also kissed horses.

May, 1920, he was taken to the Vanderbilt Clinic, where it was noted that he had involuntary movements of the head, shoulders and arms, left facial weakness, diplopia and external squint of the right eye. His gait was described as resembling that of paralysis agitans. A diagnosis of lethargic encephalitis was made.

June 11, 1920, because of the great difficulty in managing him at home, he was taken to Bellevue Hospital, psychopathic ward.

There it was noted that his general physical condition was good. The only positive neurologic sign was weakness of the external rectus of the right eye. There was no evidence of increased intracranial pressure. He was markedly overactive and talkative, distractible, unable to concentrate and fabricated freely. He aped and teased other patients and annoyed the nurses. He was very keen and observant, nothing that happened on the ward escaped him. He picked up information and knowledge quickly. He showed no lack of memory. He had no fixed ideas and gave no evidence of hallucinations. Sept. 5, 1920, his mother took him home; his mental condition was unimproved. She was, however, unable to manage him. He was restless, irritable and destructive. Kept the other children awake and could not be trusted alone. Ten days later (Sept. 15, 1920) he was returned to the psychopathic ward. He was then even more disturbed and excited than formerly. At times, he fought and screamed, spat at nurses and other patients, used obscene and profane language. He showed erotic behavior toward female nurses.

Sept. 24, 1920, he was transferred to the Manhattan State Hospital, Psychiatric Institute Service.

Physical Examination.—There were no positive neurologic findings except slight outward and upward deviation of the right eye. The gait and motor functions were normal. There were no involuntary movements. Lumbar puncture revealed 3 cells per cubic millimeter, negative globulin and Wassermann reaction negative.

Mental Status: There was marked hyperkinesis and diffusion of attention. He took an active interest in everything. He was quick in speech and motion, alert, distractible and mischievous; performed acrobatic stunts; delighted in teasing and annoying others, tried to take things from the pockets of older

patients, if prevented struck and fought or ran away and hid. He used profane and obscene language, ate and slept well, was cleanly in habits, but careless about the condition of his clothing which was always dishevelled.

His stream of mental activity showed overproductivity of speech and free elaboration, passing quickly from one topic to another. However, he answered questions quickly and to the point. When asked why he was in the hospital, he said, "I was sick and went to Bellevue—I am here because my mother couldn't take care of me." Why not? "I was raising the dickens." He then grabbed a stethoscope, put it to his ears and said "I am a doctor now." Took a cigaret from the examiner's pocket, stuck it in his mouth and said, "I am 13, I can smoke."

He said correctly that a social worker brought him to Ward's Island, that he came by trolley and boat. "Do you think I was swimming over—when I get out of here I am going to knock the sh— out of the social worker."

Emotional reaction was characterized by a feeling of well-being and elation, with quick changes to irritability and pugnacity. When asked how he felt, he replied, "Fine—I am having a good time here." "Are you afraid of anything?" "Who would I be afraid of? Do you want to fight (playfully)? You are a friend of mine, shake hands, we'll be pals."

There was no evidence of a delusional trend or hallucinations, no suspicions or fears.

Orientation was perfect for time, place and person.

Notwithstanding the overactivity and distractibility, it was possible to fix his attention with questions and secure cooperation with most of the tests. His memory was unimpaired except for the period of the acute illness (delirium) at home. His retention and immediate recall were excellent. He was able, for instance, to repeat correctly series of seven digits; he reproduced correctly nine out of ten word pair associations. Counting and calculation were in harmony with his education.

Subsequent Course.—Dec. 1, 1920, his condition was unchanged; the excitement continued.

December 15, the patient, after complaining again of seeing double, had a generalized epileptiform convulsion. Subsequently his mental state was unchanged.

CASE 12.—*Limited education and interests. Sociable, even-tempered. Good habits. Onset with eye symptoms, then lethargy. "Cogwheel" rigidity in arms. Tremors. Pyramidal tract involvement (right). Acute delirious psychosis lasting about two months. Delirious stage replaced by pathologic euphoria, associated with uncontrollable laughing attacks. "Paretic" voice. Residual tremors. Since illness, normal mentally except for the fact that she is subject to sudden unwarranted angry spells accompanied by screaming and crying and intensification of tremors.*

History.—E. P., aged 22, cigaret-maker, was admitted to Bellevue Hospital, March 11, 1920, with a negative family history. The parents and two siblings were living. She was a poorly educated factory girl. Even-tempered, sociable, with good habits and obedient to her parents. She had limited interests, was fond of moving pictures and of dancing. She was proud of the army experiences of her brother. She had had no previous serious illness.

Present Illness.—The onset was marked by eye symptoms, including diplopia, and severe head pains. After several days, drowsiness ensued and subsequently delirium. She was brought to Bellevue Hospital and owing to the delirious

state was admitted to the psychopathic ward. There she showed great restlessness and required restraint.

She was then transferred to the neurologic ward.

Examination.—On admission there, she was stuporous. Movements of the eyes to the right were limited. There was double partial ptosis, slight right facial weakness; "cogwheel" rigidity in the arms, perceptible on passive movement, also coarse tremors in the arms, with twitching movements of the facial muscles on the left side chiefly. There was more or less constant movement of the arms and legs. However, at times there was a tendency for the arms to maintain the position given them for a number of seconds. There was no hemiplegia but pyramidal tract involvement on the right. This consisted of clonus and plantar extension. The knee and Achilles' reflexes were approximately equal. There was a negative Hoffman sign. There was great respiratory distress, and breathing was accompanied by short audible inspiratory sighs, about twenty-eight to thirty to the minute.

The temperature in the hospital was 99-100 F., and at no time higher. The laboratory findings included a negative blood and negative spinal fluid Wassermann reaction, 30 cells in the spinal fluid, with positive colloidal gold series 0001232100. The spinal fluid sugar was increased.

Clinical Course.—The delirious stage lasted about eight weeks. She did not recognize persons and appeared confused. She could not cooperate, was restless and gave evidence of reacting to hallucinations. She endeavored to get from her bed and continually needed restraint.

This delirious stage gave place to a gradual return of orientation and understanding. She showed increasingly normal responses in all particulars. In respect to her mood, a real euphoria appeared. When spoken to on any conceivable subject her face broke into the broadest of smiles. Also when questioned, she said she felt very gay and happy and felt like smiling and laughing. On the other hand, she had frequent short fits of laughing which seemed to be allied to the uncontrollable laughing associated with lenticular disease. But also in these when questioned, she would say she felt happy. (There were no crying spells.) At this period the weakness of the right face was very distinctly greater during emotional expression. Because of its tremulousness and uncertainty her speaking suggested "the paretic voice." The actual distorting of speech seen in paresis was, of course, not present.

She then made steady improvement and after twelve weeks in the hospital was discharged. On leaving the hospital she showed no symptoms except a great deal of wide-spread facial tremor virtually constant and a considerable degree of the speech tremor noted above.

Subsequent Course.—When seen three months later, it was found she had been able to assume considerable housework and was returning to outside work similar to that done before her illness. She felt no incapacity for these undertakings. Her stream of thought showed no disturbances, her attitude was natural, and during the examination the mood was not unusual. It was found from her family, however, that there were alterations in mood. In contrast to a great evenness of temper prior to her illness she was now displaying anger on slight provocation at rather frequent intervals. About once a week she lost her temper, cried and screamed, did not lose consciousness and did not fall. At these times, there was great increase in the facial tremors and marked coarse jerking tremors of the arms and hand, which the patient said she could not control. These attacks were brief, lasting from only five to ten minutes. The patient appeared ashamed of them. Her parents emphasized

that they began following her illness. As regards her interests, these were meager but not different from those prior to the sickness. In a general way, moving pictures consumed all of her leisure time. There was good memory and retention and no fogginess or difficulty in thinking.

In retrospect, there was a long period of complete amnesia. She recalled coming to the hospital and the wheel chair. She had no remembrances of the first ward she was in nor of the early weeks in the second ward. She said she never felt unhappy during any of her illness. She felt no apprehension regarding death and was not anxious. In retrospect, she could not recall having had delusional ideas or hallucinations, although, as noted, the latter appeared to be present at the time. When questioned again regarding her laughing attacks, she said she felt like laughing and felt truly happy. Regarding the "spells" of a different sort which she now experienced, she said that they came because she could not control her temper as well as before. That she could not keep from screaming and crying and could not control the tremors of the arms and face. She evidenced no pride in these episodes, but instead seemed to have genuine regret concerning them.

CASE 13.—*Make-up: sociable, cheerful, optimistic, moderately alcoholic. Onset during third week of symptoms of infection (thought to be influenza with possibly pneumonia or pleurisy). Extreme weakness, diplopia and muscular twitchings in arms and legs. At first, drowsy during the day with restlessness and occupation delirium at night. Later, even during the day lacked clearness of mental grasp, was irritable and profane, complained of electricity and devil's powder burning him. After two months much clearer, then passed into a lazy, sleepy, disinterested state, with peculiar emotional reactions (drowsiness being interrupted by singing and jig dancing). Torpor and low mental tension; easily influenced when stimulated to effort by questions, then gave no evidence of impairment of mental ability. Unchanged after eleven months.*

History.—P. G., aged 39, longshoreman, was admitted to the Manhattan State Hospital, April 2, 1920. One sister in Ireland had a psychosis from which she recovered. The patient was born in Ireland, had had no serious illness in childhood, was bright in school, but only went through the fifth reader. He came to the United States twenty years ago, and had since worked in lumber yards and on the docks. He was married seventeen years ago, his wife had had five children.

About eleven years ago, he injured his back by a fall, but made a good recovery. He drank moderately during the week and was usually intoxicated Saturday night. In make-up he was cheerful and sociable, liked to sing, joke and tell comical stories. He was fond of jig dancing at home (see later under psychosis). He had had no previous attacks of nervous or mental disorder.

Present Illness.—The patient was well until the first week in January, 1920, when he complained of pain in the left side and began to cough. He continued at work for a week and drank rather heavily to cure what was thought to be influenza. He then became very weak, complained of headaches and diplopia, felt sore all over, was feverish and had chilly sensations. He spent a week in a general hospital quarantined in the influenza-pneumonia ward. He then went back home where he was drowsy during the day but delirious and restless at night. He imagined he was back at his work on the docks, constantly tried to get out of bed, thought he saw his children in the room when they were not present. There were muscular twitchings in the arms and legs.

Examination.—On Feb. 13, 1920, he was admitted to Bellevue Hospital, medical ward. His temperature was 101 F.; the pupils were unequal and reacted sluggishly to light, later they were noted as typically Argyll Robertson. Downward movements of the eyes were limited. The fundi were normal; the knee jerks, equal. Two lumbar punctures gave fluid contaminated with blood.

Clinical Course.—At first, he appeared very dull and slow of comprehension, hesitating in speech. The stuporous tendency alternated with periods of restlessness when he was more alert, showed some distractibility and flightiness in his talk. Often he was irritable and profane. He threw off the bed covers and exposed himself and urinated from the bed on the floor. He said someone was burning him up. During this period he was evidently quite confused. Finally he became so disturbed and unmanageable that transfer to the psychopathic ward was necessary on March 16, 1920. There he became quieter, but was confused and imperfectly oriented and expressed various peculiar ideas as shown when he said, "They put red pepper in my bed, it burns me all over. I sometimes feel electricity. They call it the devil's powder. It grows everywhere—blows in the window."

April 2, 1920, he was transferred to the Manhattan State Hospital with the diagnosis of lethargic encephalitis.

He was then very weak, unsteady on his feet, had coarse tremors of the tongue and hands and exaggerated knee jerks. The pupils were unequal and reacted sluggishly to light. Lumbar puncture repeated showed 4 cells per cubic millimeter and positive globulin. The Wassermann reaction was negative in both the blood and spinal fluid.

He was apathetic and inactive, had a blank facial expression, but answered questions quite readily, showing, however, considerable confusion. He thought it was April, 1921, the place a branch of Bellevue; he said he had been in the place four months (one day), that he had been in Bellevue one and one-half years altogether. Immediate retention was much impaired, he could not recall given names and numbers two minutes after hearing them. He was able, however, to give correctly considerable information about himself prior to his illness.

He had some appreciation that he had been sick; he once spoke of having been "a little touched in the head." He still asserted that he had been annoyed by devils who put fire in his bed, and he had felt electricity going through his body.

He improved steadily, and within two months his orientation and memory were clear. He realized that his ideas about red pepper, the devil and electricity were imaginations due to his illness. He continued to be rather sluggish, lacked energy, was careless about his personal appearance and generally indifferent. He remembered being taken sick at home, but recalled practically nothing about his residence in Bellevue or his transfer to Ward's Island. On June 28, 1920, he was allowed to go home with his wife.

Subsequent Course.—Aug. 12, 1920, he was returned to the hospital, his wife reporting that at home he had been very inactive and slept most of the time during the day, but at night he was restless, walking about, singing and talking. When taken out for a walk, he would playfully grab at women who passed. He would take fruit from a stand and say, "I'll pay you next time."

Nov. 16, 1920: After his return to the hospital he showed a peculiar lazy indifference and desire for sleep, interspersed with little outbursts of apparent elation when he sang and danced. He lounged about, stretched, yawned frequently and slept a great deal during the day. Very often, however, he would

rouse up without any special stimulation, seem rather cheerful and sing and dance jigs for a short time. Then he lapsed again into an inactive, drowsy state. The nurse described his behavior by saying, "He is either asleep or dancing." He was good-natured and docile, cleanly in his habits and expressed no peculiar ideas.

When questioned he was agreeable in manner, answered promptly but briefly, and had apparently no desire to go into any review of his case. The indifference in his general reaction was quite striking. He knew where he was, but was not clear as to time, thinking it was October, 1918, or 1919. He said he came here in February, 1917 (April, 1920); gave his home address correctly; seemed to answer at times in a haphazard way, for example, when asked if he had been sick, he answered in the negative. When asked why he was in a hospital, he said, "Oh, I was a little out of my head." When asked what the trouble was, he said, "I had pneumonia—it came from that." To the question as to how it affected him, he replied, "I was taken away unconscious from home—I remember nothing much about it." When asked about his present feelings and wishes, he said in a simple way, "I want to go home and get a decent bowl of tea." His wife stated that he was entirely indifferent about her circumstances. (She had to work and support the family.) He never asked her how she got along.

Questions elicited no trend of delusions, and he denied hallucinations. He had no complaints to make. As to the annoyances of which he formerly complained (pepper and electricity), he said those ideas were all imagination, due, he thinks, to the pains and soreness which he felt in his body.

Twice during the interview he got up and began to dance jigs, remarking, "I'll do a few steps of a hornpipe for you." When asked if he felt happy, he answered, "Sure." On another occasion when asked why he danced, he said it was to take the stiffness out of his limbs, and denied that he felt happy.

A special examination was made of his memory, retention, capacity to think and perform mental operations. The following is a summary of the results: There is a mild mental tension defect as shown by the errors he makes, e. g., as to time and place of an interview, his failure to register ordinary happenings about him, etc. When stimulated, however, by questions, he cooperates and is able to give, for the time being, good attention. His memory except for the period of the delirium is unimpaired. His immediate retention is good; he is able to repeat without error a series of nine digits, reproducing nine out of ten word-pair associations correctly, and in general retains what he makes an effort to. He does simple calculations promptly, more difficult ones he does slowly with ability to correct mistakes made. Orientation shows difficulty in keeping track of time, due probably to indifference in keeping informed and to lack of effort. He acknowledges no feeling of mental insufficiency.

As to his emotional state, he admitted a general lack of interest and loss of ambition. There was no essential depression. He claimed, when questioned about his feelings, that his only worry was about getting home. He did not insist on his discharge and was easily put off.

It was concluded that the patient showed essentially an indifference and mental torpor, a disinclination for mental effort, but no actual loss of capacity. The usual low mental tension and inertia could be fairly readily overcome when he was stimulated and brought to a higher level by questions.

CASE 14.— *Alert, intelligent type. Even disposition. Sociable. Extremely acute onset with head pain, then immediately a right-sided convulsion followed*

by coma lasting several days. Organic signs, facial weakness and changes in spinal fluid. No meningeal signs. Normal blood chemistry. Negative syphilitic tests. Coma succeeded by period of disorientation and confusion with dream-like ideas and hallucinations. Recovery in two weeks. Subsequent alteration in disposition. More restless. Requires more excitement.

History.—H. F., aged 21, Jewess, American born, married, housewife, admitted to New York Hospital, July 27, 1920, was one of four children. Her parents were living. The paternal grandmother was insane for a time before her death at 63, otherwise there was no history of nervous or mental disease in the family. She made a good record in common school and later in evening high school. Beginning at the age of 15 and continuing until marriage at 19, she worked as a bookkeeper in one place—a large, well-known department store. She worked with normal industry and is said to have shown more than ordinary quickness in some of the work (using an adding machine). There was a history of scarlet fever and measles in childhood. Otherwise she was never sick. Her menses had always been normal. There was no history of chorea. She was not subject to crying spells in childhood, or later, or to tantrums. She was cheerful in disposition and was always the same; she was very fond of people and of social affairs. She was not religious but attended church on holidays. Following the birth of a child in March, 1920, she developed an abscess of the breast. This caused her to become weak and nervous, and the latter showed itself chiefly when the physician, following the incision of the abscess in June, had to dress the wound daily. She would stand these ordeals very badly and would become highly upset. However, she convalesced after the abscess and on July 5 went to the country. Although she continued to be "nervous" there, in two weeks' time she gained 10 pounds in weight.

Present Illness.—We know that on July 26 she suddenly turned to her mother and said, "I've got such an awful pain in my head." Then she began crying hysterically, looked very peculiar, became unconscious and showed right-sided convulsive phenomena. That episode appears to mark the onset of the encephalitis. Five days previously, while walking with her husband, "her knees gave out" and she sank to the ground. This was transitory, and she immediately got to her feet and walked to an automobile. It is problematic whether that earlier incident should be counted as a part of the encephalitis. Following it during the five days' interval, she had absolutely no symptoms which would definitely link it with the later obvious symptoms.

At the time that she suddenly became unconscious, her right arm was drawn up and the hand strongly flexed, and her head was turned strongly to the right. These movements (apparently tonic from the account) only lasted a minute or so. The unconsciousness continued, however, and it was several days before the patient could be aroused.

In view of the later course (delirium), it is to be pointed out that the patient had not been taking any drug for sleep, that after the acute onset she was given a single hypodermic injection of morphin, and that she was received as a patient in New York Hospital in less than twenty-four hours after the onset and received no drugs there.

Physical Examination.—For two days in the hospital there was a fever of 105 F., then it was lower and reached normal after twelve days. The leukocytes were 35,000, with 83 per cent. polymorphonuclears. The chest and lungs were negative; the urine was free from pus or other pathologic findings and the blood pressure was 110. The chemistry of the blood was found to be

normal. The Wassermann reaction, in final test, was considered negative, although a weakly positive result with cholesterin antigen was first obtained. The spinal fluid on first examination showed 3 cells; later there were 20 cells. In each specimen, there was increased globulin and a positive colloidal gold curve (2233111000-1122211000) and in the second an increased sugar content (0.082 per cent.).

Neurologically, only sluggish pupils and a very slight left facial weakness (noticeable chiefly on emotion) were noted. There were no meningeal phenomena, no convulsions after the first, no change in the fundi, and no hemianopsia or pyramidal tract signs.

The patient gave variable attention. She turned a wide-eyed, childlike gaze on the examiner and displayed perplexity without fear or anxiety. There was much restlessness. She needed close watching and restraint. Both restlessness and her poor cooperation appeared due to her mood, which was one of bewilderment linked with something suggesting mild euphoria. She was disoriented in all the spheres. Her speech was quite limited in amount, somewhat but not markedly irrelevant, and not incoherent. Some days after her first visit, she failed to recognize the examiner. Hallucinations, probably illusory, were present. She thought the nurses said "Ferndale" (where she had been) over the telephone. Delirious ideas were not expressed to the examiner, but they were present, as the following shows: From her husband we find that she confused him with her brother and when they both came to see her, said she was married to the brother. Another time she said, "I am married twice, I can't think of the other man but I have two children—ain't I crazy?" On one occasion she wanted to be allowed to have two eggs in the bed with her. Then (although this request had not been granted), she threw all the bedclothes off and called to a nearby patient to notice that the eggs were not touching her—she also said that there was nobody in bed with her.

From talks with her, following recovery, one finds that she imagined there was a small fat man under her pillow. (No idea regarding his being Lilliputian. Although he kept under her pillow he was a real-sized man.) Also she mistook the electric light bulbs for men. She would watch them "take off their heads, then their arms, and then they would disappear down the pipes" (chandelier).

A neurologic symptom, not conspicuous at the time because of her mental state, but which she described afterward, was a sight disturbance, a certain blurring of vision. Her eyesight seemed unnatural for a while, but there was no diplopia at any time. Her acute mental symptoms lasted about one week and she was able to leave the hospital after fourteen days.

Subsequent Course.—When interviewed twelve weeks after her hospital stay, it was found that her family considered her more restless than before her illness and less settled. She herself said she could not stand staying home and that she wanted excitement. She went to some theater almost every day. Otherwise, there seemed to be no change in her. She was not neglectful of her child in spite of her desire to be out. She had her usual grasp on affairs and was, for her training, properly interested in events and in her friends. The memory was good and retention was excellent. She attributed a difficulty in sleeping to the fact that at night she frequently needed to look after her young child.

CASE 15.—*Emotional instability indicated in make-up. Onset five weeks after influenza with meningeal symptoms. Pain in head and neck, vomiting and diplopia. High fever and somnolent state for a few days, followed by*

hallucinatory delirium. As sensorium cleared, she was for a short time in a depressive apprehensive state, feared she was to be tortured and killed, that an abortion was to be performed, etc. Rapid recovery in three months with amnesia period, but recollection of much of the mental content during the delirium. Remains well one and one-half years after discharge.

History.—E. G., aged 26, married, wife of bookkeeper, was admitted to Manhattan State Hospital, May 3, 1919, with a family history that was negative for nervous or mental diseases. The patient was born in New York City and graduated from grammar school at the age of 14. She was ambitious and intelligent; lively and happy in disposition, at times perhaps "too happy." She made good wages in clerical positions. She was married in January, 1918, and three months later her husband and also her brother joined the army. As a result of this separation, she felt quite downhearted and blue. She soon appeared cheerful and happy again, in fact, became rather elated, but told her friends that she did not know why she should seem so happy when in reality she was sad about the absence of her husband. (The statements regarding her make-up and the reaction to separation from her husband suggests quite strongly a cyclothymic constitution.)

Present Illness.—Jan. 25, 1919, she became ill with influenza and was treated for five weeks in Bellevue Hospital. She left the hospital apparently recovered except for general weakness and some trouble with her bladder, frequent and painful micturition, which had followed the influenza. Four days after returning home, she was again taken acutely sick with pain in the occiput, stiffness of the neck, shooting pains in the back and vomiting. Three days later, she was taken to the New York Hospital (March 8, 1919).

She was then dull and somnolent but could be aroused. Her temperature was 104 F. Lumbar puncture revealed clear fluid, 30 cells per cubic millimeter, negative Wassermann reaction, slight reduction of Fehling's solution. There were exaggerated deep reflexes, and a bilateral Kernig sign. She complained of pain in the head and neck and double vision. The urine was negative, but a week later pus cells were found in a catheterized specimen. The fever subsided and the temperature was normal the fifth day after admission. Coincident with this, the somnolent tendency was succeeded by a restless, noisy, talkative state, with evidences of a clouded sensorium. She misinterpreted her surroundings, talked disconnectedly, "used meaningless words," mumbled and "gave pseudo reminiscences." She became more disturbed and difficult to manage and made attempts to get out of her window. Her case was reported to the board of health as a case of lethargic encephalitis, complicated with psychosis. April 2, 1919, she was transferred to Bellevue Hospital, psychopathic ward.

The patient was in the Psychopathic Ward from April 2 to May 3, 1919. There she was described as being in a depressive, apprehensive state, hallucinatory, restless and disoriented. Profuse crying is said to have been a prominent symptom. Her utterances which show her confused state of mind and reaction to hallucinations are illustrated by this: "I see them passing by—there she is now—I see her doing something with a whip—she was twirling it at me—they want to whip me—listen to the marble falling down—I don't know why the people dislike me—Now what is that, who hit me on the side of the head—God tried to get rid of me." She expressed the idea that a criminal abortion was to be performed on her.

From Bellevue she was sent on May 3, 1919, to the Manhattan State Hospital. She was then in an excited, hallucinatory state. She asserted that she

saw axes, knives and flames and thought she was to be destroyed. She called out, "Fire—fire," and frightened other patients. She heard voices say she was to be tortured and killed. She denounced and cursed her husband when he visited her. The following day she was much quieter. She mistook the physician for a former acquaintance. She knew she was on Ward's Island, realized she had been sick and asked about going home.

Physical Examination.—At this time she had active knee jerks. Other wise the examination was negative for neurologic signs. There was some pulmonary congestion of both lower lobes. She was poorly nourished. The temperature for two weeks ranged from 99 to 100 F., twice going to 101.

Clinical Course.—From this time on, the mental condition rapidly improved. The fears and hallucinations disappeared, she became clearly oriented and generally cheerful in mood, with, however, some tendency to fret over being in a hospital for mental diseases, and also to cry if her husband missed coming to visit her.

By May 19, 1919, she was still very weak physically, but was regarded as having practically returned to a normal mental condition except for some emotional instability shown by weeping easily when talking of her illness and the mental distress which she went through. She seemed quite anxious to forget the thought that she had been mentally deranged, and hoped that she would transmit no taint to her offspring if she should have any. A review of her early life, personal data, school knowledge and general memory gave no evidence of any mental impairment. Her retention was excellent, thinking capacity good and insight satisfactory.

Subsequent Course.—The retrospective account of her illness showed that she was able to recall her symptoms very well until shortly after the lumbar puncture at the New York Hospital. About that time, she became confused and recalled little of what happened about her. She remembered, however, a good deal of the mental trend, including the delirious ideas and hallucinations present during the period of confusion. She thought many people were dying, it seemed that every patient put in a bed next to her died. She thought some of those who died were relatives. At times she imagined she was at the war front and saw soldiers going over the top. (Prior to her illness she had worried much about her brother and husband, both of whom were in the army.) She recalled the abortion fancy and connected it with the bladder trouble which followed the influenza and the suggestion that she might require a surgical operation. Her fear of fire was explained as being due to a dread of fire since childhood, when her mother was burned to death.

She remembered nothing of the transfer to Bellevue or her residence there. She recalled, however, coming to Ward's Island on a boat, but she thought she was on her way to Europe. After this she gradually became clear as to her whereabouts.

June 5, 1919, she left the hospital recovered from the psychosis. She spent some weeks in the country, where she rapidly gained in weight and physical strength. She then returned to her home. July, 1920, she gave birth to a healthy child.

Nov. 17, 1920, she was reported as well both mentally and physically.

CASE 16.—*Quiet, sensitive and rather seclusive make-up. Oculolethargic onset with two months of mild stupor and confusion. This was followed by general weakness, leg pains and disagreeable twitching of tongue. Prolonged depressive, complaining emotional reaction and dissatisfaction with hospital treatment. Then in a boarding house developed a paranoid trend with sus-*

picions, delusion of poisoning and auditory hallucinations, called bad names, etc. On reentering hospital no further elaboration of paranoid ideas, but has gained no insight. Physical complaints and mild depression continue after lapse of two years.

History.—L. M., aged 42, single, seamstress, was admitted to the Brooklyn State Hospital, Feb. 17, 1920, with a family history that was negative for nervous or mental disease. She was a native of New York. She was delicate as a child and obtained a common school education. In disposition, she had always been quiet, sensitive and worrisome. She had lived a rather secluded life, making her home until recently with a maiden sister, both working as dressmakers. Seven years ago, she was successfully operated on for cancer of the right breast. There had been no previous attacks of nervous or mental trouble.

Present Illness.—About Jan. 1, 1919, she developed weakness and nausea, symptoms that were thought to indicate influenza. Within a few days, she became stuporous and slept most of the time for two months. She could be easily roused but would quickly fall asleep again. It is doubtful whether she showed any delirium, although she sometimes muttered in her lethargic state. She expressed no delusions. Her eyes felt as if they were stiff and not easily moved, and she had some difficulty in vision at this time.

Feb. 25, 1919, she was admitted to the Neurological Institute in a stuporous condition. She then had irregular, sluggish pupils and exaggerated knee jerks. Lumbar puncture revealed 6 cells per cubic millimeter. The Wassermann reaction was negative. She gradually recovered from the stupor and was discharged at the end of one month with the diagnosis of "encephalitis lethargica." Subsequently, she complained of general weakness, severe pains in the legs, and a peculiar disagreeable twitching of the tongue. Because of these symptoms, she was treated in several different hospitals. During this period, she seemed to have been quite despondent, cried a good deal, complained constantly of leg pains, and probably did not adapt herself well to hospital routine or cooperate in the treatment proposed.

While in St. Peter's Hospital, Brooklyn, during the fall of 1919, she became suspicious that the medicine was harmful and thought people were talking about her. She complained of pains and aches and wept almost continually. Because of her peculiar behavior, she was sent to the Kings County Psychopathic Ward, Nov. 29, 1919. She was then depressed, moaned and cried, spoke of the pains in her legs, a bitter taste in the mouth and a "quivering" of the tongue. She denied any paranoid trend. After two days her sister took her out and placed her in a boarding house.

She then developed a definite paranoid trend against the landlady who took care of her. She asserted that the medicine was poison, that "dope" was put in the food. She said the landlady was trying to prevent her from leaving to go to a hospital for treatment, that she wanted to keep her because she needed the board money. She said she overheard remarks made by the landlady and her son; they called her bad names and made slurring remarks, for example, "She is no good; she is a bum, a hypocrite. Her people do not care for her." She also heard something mentioned about the "sin of impurity."

Because of her trend against the landlady, her sister had to take her back to the Kings County Psychopathic Ward in February, 1920, and she was then committed to the Brooklyn State Hospital.

Physical Examination.—At this time she showed left external strabismus, moderate ptosis of the left lid, with a history of diplopia; weakness of the left side of face; right pupil larger than left, both limited and sluggish in reaction to light. There were coarse tremors of the tongue; the knee jerks were increased. Lumbar puncture revealed 4 cells per cubic millimeter, the Wassermann reaction was negative for both fluid and blood.

The mental status showed that the patient was composed and tractable. She was worried about her present situation, a little suspicious about the writing up of her history and ready to combat any suggestion that she was insane. She was clearly oriented and gave correctly her personal data. Her memory was unimpaired, except for the period of the stupor; retention was good. Simple calculations were performed promptly and accurately. She told of having influenza and sleeping two months, she said she remembered little of what happened during that time. She had subsequently suffered much from pain in the legs and from general weakness. At some of the hospitals, they did not seem to want her. The main trouble was with the landlady where she last boarded. She put something in her food, it made her vomit up bitter stuff. She also called her bad names.

Clinical Course.—Following her admission to the state hospital, the paranoid trend was not further elaborated. She continued to complain of her leg pains, could not be interested in any occupation, spent most of her time on the settees or the bed, and showed no somnolence.

Nov. 17, 1920, the patient was still in the hospital. She remained in bed because of her leg pains. She was accessible and able to give a good account of her illness and previous history. Orientation was correct, memory showed no impairment and retention was good. She was alert, attentive and responded promptly. Apperception and mental elaboration were not apparently interfered with. The emotional tone was one of mild depression and worry over her condition. This should perhaps not be considered pathologic, as she was bedridden and suffered much, so she claimed, with pains in the legs and a disagreeable twitching of her tongue. Several times during the interview, her eyes filled with tears when she spoke of her trouble and present situation.

She recalled well the onset of her sickness. She said that she had "influenza" followed by a stuporous period for two months, during which time she knew little of what went on about her. She was taken to the Neurological Institute where she improved, brightened up, but was troubled by pains in the legs and a peculiar twitching of the tongue. She was then sent to a convalescent home and later to several other hospitals. She denied that she had any suspicions or that she made any complaints of mistreatment in any of the hospitals, but admitted that in the boarding house she became suspicious and expressed the idea that the landlady was "doping her." She was dissatisfied with the place and thought the landlady was giving her quieting medicine so as to keep her from leaving; she thought the landlady needed the board money, this was the object in drugging her. She asserted that she had no suspicions or cause for complaint about her treatment since leaving the boarding house. She still believed, however, that the landlady did drug her and did make remarks about her.

There was outward and slightly upward deviation of the left eyeball. Knee jerks were equal and active. Plantar stimulation caused flexion of the toes. She complained of pain in the legs below the knee only. She lay with her feet in the position of hyperextension (suggesting foot drop). When tested, however, there was no weakness in the extensor muscles. The calf muscles

were tender to firm pressure. The nerve trunks were not abnormally sensitive. The gait was rather stiff. She could, however, walk and turn without support. There was no Romberg sign.

The tongue showed no atrophy, no marked tremor and no distinct deviation. There was slight weakness of the left side of face.

Dec. 20, 1920, her memory retention and thinking capacity were tested in detail. She showed slight mental tension defect but no impairment of the intellectual faculties. Of late, the patient had been again a little drowsy, with less interest in her surroundings. Emotional instability and depressive affect continued.

CASE 17.—*Good make-up, friendly, sociable, efficient. Acute onset with fever and oculolethargic symptoms. Stupor for forty-five days, during which she developed weakness of the left side of body. Following stupor, depressive, complaining, discouraged attitude based on her illness (continued weakness and partial disability from paralysis). After eight months severe depressive psychosis with hallucinations; fear of being killed, attempts at suicide. Later peculiar emotional reaction, smiling and talking of being killed. After fourteen months continues depressed with suicidal inclinations and contradictory mood reactions. Little evidence of any organic impairment of mental faculties.*

History.—D. M., aged 29, Russian Jewess, wife of a carpenter, was admitted to the Manhattan State Hospital, May 24, 1920. She had been strong and healthy as a child. She came to the United States at the age of 18. She had little opportunity to go to school, but was bright and intelligent. She was an embroidery worker and earned good wages. She was married at the age of 20, was happy and contented in her home life and had had three children. She was friendly and sociable in disposition, ready to laugh and enjoyed amusements. She adapted herself well to circumstances and was not easily discouraged. There had been no previous attacks of nervous or mental disease.

Present Illness.—The last week in September, 1919, the patient complained of headache, pain in the back and sore throat. She continued, however, to do her work until October 1, although she was feverish and somewhat restless. On October 2, she complained that something was wrong with her eyesight and that she saw double. She went to bed and then passed into a stuporous condition, during which she kept her eyes closed and could be aroused only with difficulty. Her temperature during the first few days of the stupor ranged from 103 to 104 F. On Oct. 16, 1919, she was admitted to the Willard Parker Hospital. The report from that hospital states that she was in a stuporous condition on admission, with slight rigidity in the limbs and a tendency to keep them in given positions (catalepsy). The pupils contracted. Knee jerks were sluggish, plantar reflexes were normal. No Kernig sign or rigidity of neck was elicited. There was no evidence of paralysis. The patient muttered a few words while being examined, but could not be aroused. She picked at the bed clothes.

Lumbar puncture gave a clear fluid with 20 cells per cubic millimeter. The culture was negative. The Wassermann reaction was negative for both blood and fluid.

The patient remained in a stupor with her eyes closed for about forty-five days. She could always be partially aroused by shaking, then she usually muttered, but rarely said anything which could be understood. Toward the last of November, it was noted that there was weakness on the left side (face and limbs). She also developed large bedsores.

She came out of the stupor about December 1, began to talk and quickly improved so that she could sit up. She had trouble in using the left arm and leg and complained a great deal of headache and pain in the legs. The bedsores did not heal, she became discouraged, dissatisfied and restless, and talked of being a permanent cripple. March 22, 1920, she was transferred to Bellevue Hospital (medical ward) with the diagnosis of lethargic encephalitis.

At Bellevue, she cried and complained of pain, was generally dissatisfied and begged to be taken home. She was taken home April 1, 1920. She then seemed more contented and in two weeks was able to be out of bed. She was weak, but not sleepy. She spoke about her "lazy" condition and wondered why her husband did not scold her because she was "good for nothing."

May 1, 1920, a sudden change occurred which marked the onset of pronounced psychotic symptoms. She began to talk of her relatives and husband wishing to kill her; she became agitated and restless with marked suicidal tendencies. She said she did not wish to live, she had too much trouble. She tried to jump from the window, poured boiling water on her head, drank a bottle of medicine in the hope that it was poison. She did not accuse herself of wrong doing, the main idea was that she should die on account of being sick and partially disabled.

She was returned to Bellevue Hospital and placed in the psychopathic ward. There she was anxious and apprehensive, would strike others and said the children were destroyed. The orientation was approximately correct; there were no signs of delirium. She began to show a peculiar emotional reaction. When visited by her husband, he noted that she laughed a good deal even while she talked of her children being dead. Although laughing, she did not seem happy, in fact, just the opposite.

May 24, 1920, the patient was transferred from Bellevue to the Manhattan State Hospital. She appeared very depressed, responded in a low voice, with little change of facial expression. She was oriented and asked to be killed, adding that she had poured scalding water on herself, and remarked, "I broke the Jewish temple."

Examination.—Physically, there were left hemiplegic residuals, with weakness of the face and some spasticity of the arm and leg. She walked on the ball of the left foot (talipes equinus). Knee jerks were both increased; no Babinski sign was elicited. Lumbar puncture revealed clear fluid and no cells. The Wassermann reaction was negative for both blood and fluid.

July 27, 1920, determination of a complete mental status was attempted, but the patient gave very poor cooperation. She replied indifferently to questions or showed annoyance and disinclination to answer, yet at the same time she smiled frequently. A few of her responses will indicate her attitude: Do you feel sad? "Don't speak to me." (Smiles.) Do you feel happy? "Don't speak to me—yes, I do." Treated unkindly? "No. Don't speak to me." (Smiles.) Hear voices? "By the mountain—I don't want you to speak to me."

It was not possible to ascertain anything as to the mental trend or to determine the presence or absence of hallucinations.

She was fairly clear as to time, but said she was in the Lebanon Hospital. She misidentified the other patients, also the physician. No estimate could be made of her general memory or of what she recollected of her sickness. Her stock answer was simply, "I don't know," without appearing to make any effort to give information.

Clinical Course.—During September, 1920, it was noted that in some respects the patient had improved. She was able to be up and was not so indifferent

and averse as formerly. She spoke of wanting to get well and to go home. She began to help a little with the ward work and showed that her memory was at least fair, as she had learned the names of all the patients on her ward. She was more friendly; in fact, she showed some inclination to playfulness and to tease the others. When asked the name of the place, she said "a church," but it is doubtful whether she was serious. To the physician she said, "I wish you no luck—you are going to kill me." It was still impossible to examine her satisfactorily about her illness.

Nov. 30, 1920: At several interviews lately it was found that the patient was more accessible than formerly and in some ways improved. She showed more interest, made more inquiries about things at home, helped the nurse about the ward and was attentive to some of the feebler patients. She had given up the idea that her children had been killed and did not mistake identity as formerly. There were no peculiarities of conduct.

Her general attitude and emotional state are difficult to analyze. She showed a peculiar mixture of suspicion and uneasiness, with harping, during the interview, on the idea that she was to be killed by the physician, yet she was compliant, would smile on suggestion, seemed momentarily friendly and even laughed a little. Then the mood changed quickly and she accused the physician of wanting to murder her, and seemed fearful and wanted to leave the room. At other times, she would declare, "You want to kill me," without any special show of affect; in fact, she could often be made to follow her declaration with a smile in response to the physician's smiling or joking with her.

There was a distinct depressive paranoid trend with hallucinations or misinterpretations of remarks of others. When asked why the physician or anyone should wish to kill her she said, "I insulted the whole crowd. My husband didn't pay money to you for the food." She heard this yelled out "by Christian men around the building." They also called her names as "Yiddisher colver —Yiddisher bad woman." She denied that she had done anything wrong, but at Bellevue they hollered out that she "broke the Jewish temple."

She admitted that at home she wished to die because she was nervous and sick. She thought that when she poured hot water on her head her children died. She acknowledged this was imagination.

When asked to explain why she smiled and talked of being killed she said, "I don't know myself—it is foolish to smile—I am unlucky."

She was now clear as to her surroundings but sometimes failed to give the date accurately. In general, her memory was good but there was some evidence of a mild mental tension defect as shown by the variability of the accuracy with which she recalled and correlated past events at different examinations. For example, on one occasion she made several errors in giving the birthdays of her children and was a little mixed up about the date of her marriage, whereas at another interview she gave these data correctly. When she could be induced to make an effort she showed good immediate retention. She made some mistakes in mental calculations but could correct these. On the whole, she did quite well. She herself, however, felt some loss of mental capacity, which was expressed by saying that she got mixed up easily and could not count as quickly as before her illness. (Formerly she had a very good head for business.)

In conclusion, it can be said that there were only slight evidences of an organic impairment of the faculties; a mild mental tension defect with some variability in capacity, but ability to correct any errors made. Retention was

good, when cooperation and interest were secured. A most striking symptom was the peculiar mood reaction which superficially at least suggested a lack of correspondence between the affect and the ideas expressed. We do not believe, however, this reaction was similar to that of dementia praecox. It was rather the quick change of mood that is misleading, and when the patient talked of being killed she seemed actually afraid. The smiling, however, seemed beyond her control and was certainly easily elicited on suggestion. She herself had characterized the reaction by saying, "I know it is foolish to smile." Also the underlying genuine affect was shown by her repeated attempts at suicide.

Dec. 7, 1920: The patient recently made another serious attempt at suicide by hanging. This was preceded by exacerbation of the depression and fear of being killed.

CASE 18.—*Well educated school teacher. Social, athletic, intellectual type, probably over-religious and superficial in interests. Rejection of marriage on grounds directly dependent on this religious side of her character. Later regret and increasing bitterness. Began to turn against religion. Developed ideas concerning injustice of God. No peculiarities of conduct. Continued efficient at her work. Epidemic encephalitis for twelve weeks. Stupor and occupation delirium. Good recovery. Since, strange beliefs concerning the magical and remarkable results of her illness. Elaboration of lengthy religious ideas including the belief that the second Christ, to come, is to be a physician. Also believes law should be passed to make birth of children illegal. Has prepared manuscript setting forth views against God, whom she regards as a murderer and perpetrator of evil. Does not express these ideas openly in ordinary conversation, but is easily drawn out to discuss them. Continues efficient at teaching. Case considered schizophrenia, intensified by the recent brain disease in an individual with former tendencies in that direction.*

History.—L. B., aged 31, teacher (domestic science), was admitted to St. Luke's Hospital Jan. 20, 1920. She was one of a large family; her parents were living. There was no history of nervous or mental disease in the family. She had had measles, chickenpox, scarlet fever and whooping cough in childhood, and at 23 otitis media, complicated by a mastoiditis, and soon afterward pneumonia and empyema, from all of which she is said to have made a good recovery. She was brought up in a substantial rural community, in a large family, where religious and, to some degree, cultural things were emphasized. In make-up, she was described as having always been both social and athletic. She was especially fond of, and clever at, winter sports. Although very much interested in intellectual matters, she was not bookish. Instead she was fond of many people and popular among them. She continually improved her knowledge for her teaching, usually taking a summer course each year. Her friend said she was known to be a good teacher. She was clever at making things, being "generally handy," yet she never took as great pride and interest in such accomplishments as her skill would have warranted. This appears to indicate a certain superficiality of interest.

A few years prior to her illness, she became acquainted with a young man of less education than herself, who, however, was making a good living in a business. Though "going straight" at the time, he had a record for excessive drinking and "came of a poor inheritance" (inasmuch as his parents had been unable to live together). He was not at all religious and was inclined to be a ready spender of money easily made. The patient, when it came to the point of a choice between an engagement or breaking the friendship, chose

the latter because of the man's "inheritance." This her friend said she did because of her family, and yet she did not actually counsel with them about it. And also as soon as she had made the decision, she became very regretful of her choice. Incidentally, the man very shortly was out of her reach. A considerable bitterness toward life then developed, and although the friend could think of no definite things which the patient said at that time, she said that "stray remarks," beginning then, showed that the patient at that time began to turn against religion and against the ideas which she had formerly fervently held.

Present Illness.—Jan 20, 1920, she entered St. Luke's Hospital, complaining of dizziness, nausea, vomiting and diplopia, and with the following history: Eight days before admission, she awoke with a dizzy feeling. She vomited a moderate amount, three times. The nausea was relieved, but the dizziness kept up. She taught school that day. The next day she awoke with diplopia, which caused more dizziness and nausea. She got through that day's work by keeping one eye closed all the time. Then she spent from the fifth to the eighth days of her illness at home in bed "trying to sleep the trouble away."

Examination.—In the hospital this showed slight intention tremor of the hands, tremor of the tongue on extension and no ataxia in finger to note tests. There was no Romberg sign and no paralyses. Biceps, triceps, wrist, knee and Achilles' reflexes were present, equal and normal. No Babinski or Kernig sign was elicited. There was pseudo ankle clonus on both sides. The gait was that of weakness. On admission the fever was 103 F. After eight days it returned to normal, then rose again to 101 on the thirty-third day, and for a six or seven-day period subsequently was subnormal, even 96.6 on three occasions and once 96. The spinal fluid showed 21 cells, sterile culture, a negative Wassermann reaction and a colloidal gold curve of 122210000. The leukocytes were 15,700, 76 per cent. polymorphonuclears.

Clinical Course.—The following notes show her condition in the hospital. On the second day, "the patient rouses when spoken to, otherwise appears to sleep, mumbles and talks to herself." "There is general resistance to passive motion in arms and legs." Two days later, "still shows catatonia. Talks rationally when aroused." The day after the temperature became normal (and for six days it had been only 100) the note was made, "restless and talkative during night and today. She was delirious, did not know visitors and persons of the hospital and a great deal of the time talked to imaginary people as if she were at school and teaching." Her delirium was almost entirely occupational. There was a gradual subsidence of the symptomatology and after eleven weeks in the hospital she was discharged, having reached a safely convalescent condition. She went to her home in Pennsylvania by train (wheeled chair).

Subsequent Course.—The great interest in this case lies in the mental condition which the patient was found to show when interviewed after an interval of six months (October, 1920). The most striking features were an elevation of mood and an unusual productivity of speech, with the expression of a peculiar trend of ideas. The elation showed itself not alone in the content of her thought and by a great volubility, not accompanied by a flight of ideas, but also by an unusual and somewhat indecorous abandon of attitude.

She said, "I have no longer the power to suffer. I have no tears." Her speech was rapid, fluent and insistent. She brooked interruption but preferred

to keep talking. She evidenced considerable quickness of thought. Her speech was relevant—to a very slight extent incoherent. At least in regard to a creed which will be discussed later, it was ecstatic and hazy. Frequently she reverted to expressions telling how happy she now was. She was not distractible.

She said that the sleeping sickness had worked a marvelous change in her, that it had made her over, that before it her great sin had been worry and that the illness has taken "worry" from her. In fact, she remarked, "My worry center is dead." She believed that the lumbar punctures in the hospital wrought her magical cure. She knew that the encephalitis took sin (worry) from her and asked what rôle the physician had in it and also which physician it was. She expressed the feeling that there must be many people with worry as their sin, who, if they could only have the "sleeping sickness," would be made well. She felt it to be her mission (though she did not emphasize the crusade idea at all) to see that some physician found these people and inoculated them. Along with these ideas, she had formulated her antireligious ideas and recited them as her creed. This was a series of long, high-sounding phrases of religious coloring, of great length, pronounced with astonishing rapidity, clearness of enunciation and earnest fervor. Later, much the same, in less studied form, was written down and reads as follows: "My faith, in brief, is as follows: I don't believe in the existence of a God, hence prayer is futile. I don't believe in a life beyond the grave, death ends all. I don't believe in the Bible. It is the most inconsistent book that was ever written. I am happy in this belief, for I do believe, and firmly, that God was human, not divine. Possessing superhuman power, he made this wonderful earth and sky and sea which are the foundations of Heaven. Then he inhabited it with beings but left us, their outgrowth, living in Hell, for he died before his work was perfected. . . . Now I believe just as firmly in the coming of a second Christ who will perfect Creation. As the work that needs perfecting is of a medical nature, that second Christ will be a doctor, also human but exhibiting supernatural powers." She completely filled 109 pages of notebook stationery with a discussion of this topic. It is presumably an autobiographic account of her religious life, but both at the beginning and at the end, and repeated innumerable times in between, these ideas are put down along with unnumbered tirades against the injustice of "the God whom she was taught to worship. Not only is he a murderer of millions, but he is millions and millions times worse than a murderer when he hands down to innocent children the sins of the fathers." Also "when the kaiser claimed to be in league with God during the World War, were you surprised? Not I, for I believe the proverb, "Birds of a feather flock together.'" Later, on a different theme, "As the world is today, children are brought into it by the devil passion. I want to live to see the day when this powerful devil will be knocked out of existence, and common sense, the Christ, will prevail . . . until that time comes I would like to see a law passed forbidding marriages and births, a law requiring the segregation of the sexes if necessary . . . I suggest that man step in and bring it (this world) to an end."

The patient is said not to mention these ideas about God unless they are brought up. Her ideas regarding her magical cure from worry are more superficial. And she has made a point of quizzing physicians and others, not concealing her more than strange faith in the miracle the encephalitis worked in her.

Her friend believes that she is more composed and more contented than prior to her illness and more inclined than before to stay home quietly evenings and read.

There was no impairment of memory or retention. She was teaching successfully. She showed poor judgment regarding many of her present ideas. On the other hand, she manifested complete insight into the delirious experiences in the hospital. There was an amnesic period during the first part of her illness in the hospital. Then she told, with good insight, of such delusions as the following which she had: The occupational delirium has already been mentioned. She mistook the visiting rector for St. Peter. She knew she was no longer on earth, and knew that she was in Heaven. This gave her a feeling of great joy and satisfaction. She did not understand why they should take her to "Jersey" from the hospital. She was puzzled by the idea of there being a "Jersey" in Heaven. She said it took her two weeks at home in Pennsylvania to realize the true state of affairs. "After two weeks I suddenly realized that I was not in Heaven but back in this Hell of a world again."

In her account, she wrote, "In the hospital I thought I was being prepared for Heaven. Every body massage, every salt water bath, every alcohol rub, every visit of the doctor, were all done in preparation for my Heavenly home."

This 109 page article is entitled "Praise God and Stop Worshiping the Devil, Written by a Victim of the Devil and Dedicated to Those Who Need It Most, viz., Ministers, Priests, Rabbis, Missionaries, etc."

More recently she has complained of a catarrhal condition of her nose and throat and has seen several physicians for this, none of whom has been able to find anything wrong. At the same time, she complains of her saliva and considers that it is thickened and has turned white. She spends about thirty minutes each morning clearing out her nose and throat, and at later intervals during the day goes through similar prolonged procedures for this purpose. Her roommate complains that she is continually spitting in an annoying fashion. She has shown no tendency to violate usual proprieties regarding this. Her friend considers these recent ideas on this subject rather "foolish." A less intimate girl friend of the patient, having in mind the religious disbeliefs and ideas concerning her rejuvenation by means of her sickness, made no greater comment than, "My, she has a weird philosophy." In other words, there is casually observed very little unusual behavior. It is impossible to establish that she is abnormally secretive, suspicious, etc.

HISTOPATHOLOGIC FINDINGS IN A CASE OF SUPERIOR AND INFERIOR POLIENCEPHALITIS WITH REMARKS ON THE CEREBROSPINAL FLUID*

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CHICAGO

REPORT OF CASE

The patient, a man, 21 years of age, was admitted on Aug. 20, 1920, to the neurologic service of Cook County Hospital complaining of headache and vomiting, with inability to swallow and to judge distance.

Previous History.—There was nothing abnormal noticed in the history of the patient's childhood except poor vision and "inability to judge distance." At the age of 9 he went to school, but "could not learn fast" because of defective vision. He was infantile in his desires and preferred to play with babies and young children.

About six months before admission to the hospital he became dizzy and fell downstairs. He was picked up uninjured, but remembered nothing about the accident.

He began to complain of his present symptoms in the early part of July, 1920. The most distressing symptom was inability to swallow, which had been present for about six weeks. For two days after admission the patient was able to swallow only small amounts of liquids. During the same period of time speech became quite indistinct and almost unintelligible.

Headaches had been present for the last two years, mostly frontal in distribution. They had been coming in attacks which lasted for days or weeks and then spontaneously disappeared for two or three weeks. For the past six weeks vomiting without nausea would set in after each meal. During the six weeks prior to admission there was a marked increase in the strabismus which had been present, in a mild form, since birth.

Previous Illnesses.—Measles, whooping cough, mumps; he had had no operations and had received no injuries.

Family History.—The patient had five brothers and one sister living and well. One sister was not bright mentally; otherwise the family history was negative.

Examination.—The appearance of the patient was that of a not well-nourished boy, dull expression and defective mentality. He gave inadequate answers even to the simplest questions, though he was well oriented as to time and surroundings. There were no visible paralyses of the spinal nerves, but almost all the

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* From the pathology laboratories of Cook County Hospital and Illinois State Psychopathic Institute.

* Read at a joint meeting of the Chicago Neurological and Ophthalmological Societies, Chicago, Dec. 16, 1920.

cranial nerves were involved. Thus, the eyelids were drooping, especially the left; the eyeballs were almost totally immovable, only slight movements up and down being possible. The palpebral fissures were greatly narrowed, especially the left, which was represented by a narrow slit. The facial muscles were immobile, wrinkling of the forehead, widening of the mouth and puckering of the lips being almost impossible. The mastication muscles were markedly weak, the uvula and soft palate immobile, and swallowing even of small amounts of water was greatly impaired. In fact, the patient did not even attempt to swallow, as the food would "stick in the throat" and the liquids regurgitate through the nose. The tongue could not be protruded beyond the teeth; it showed twitchings and some atrophy but no deviation.

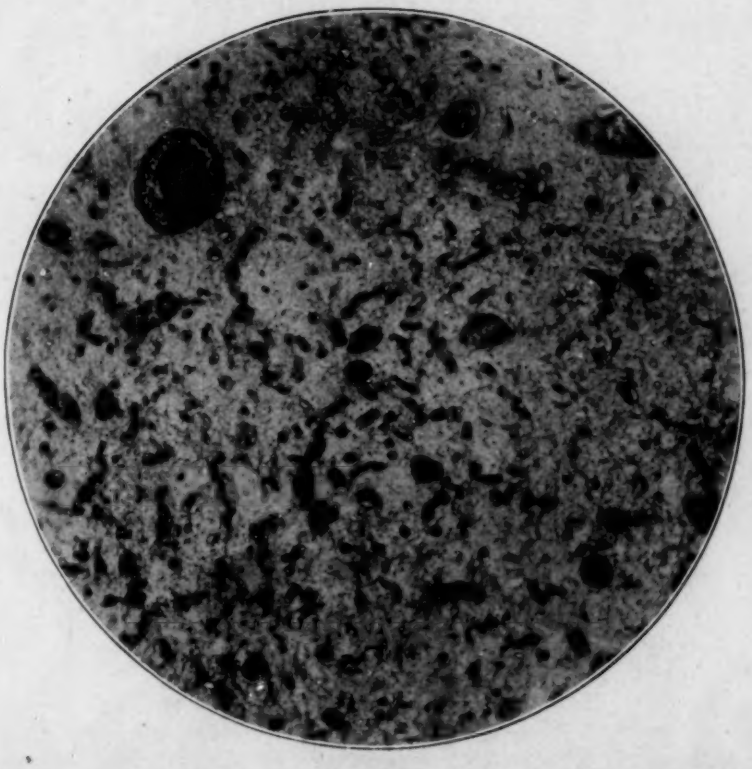

Fig. 1.—Tegmental portion of the pons. The entire area is covered by a great number of vessels and capillaries, represented in the illustration by dark, round bodies of various sizes. Some are so small that they can be seen only with the help of a hand lens as minute capillaries. The glia is vacuolated and covered by glia nuclei, ganglion cells and other structures photographed separately. Alzheimer-Mann stain; × 65.

Reflexes: Both pupils reacted to light; the corneal, conjunctival and pharyngeal reflexes were greatly diminished; the triceps, knee and ankle jerks were weak; the abdominal and plantar reflexes were normal; Babinski sign and ankle clonus were absent. Sensibility was normal. The muscle power in the extremities was fair; the deltoid, supraspinati and infraspinati and pectoral

muscles were somewhat atrophied. The heart was slightly enlarged, the right limits reaching the right border of the sternum, the left limits reaching one inch beyond the left of the nipple line; the apex beat was palpable in the fifth intercostal space; there was a distinct systolic murmur. Examination of the chest revealed scattered râles on the right side, dulness over the apexes with bronchial breathing and a friction rub over the lower right lobe. Respiration was 40, the pulse rate 84 and the temperature 99 F. (by rectum).

The abdominal organs showed no abnormalities. The spinal fluid was under normal tension, colorless, with 5 lymphocytes per cubic millimeter and a negative Nonne test. More detailed examination of the patient's mental and physical condition was impossible as he died suddenly two days after admission.

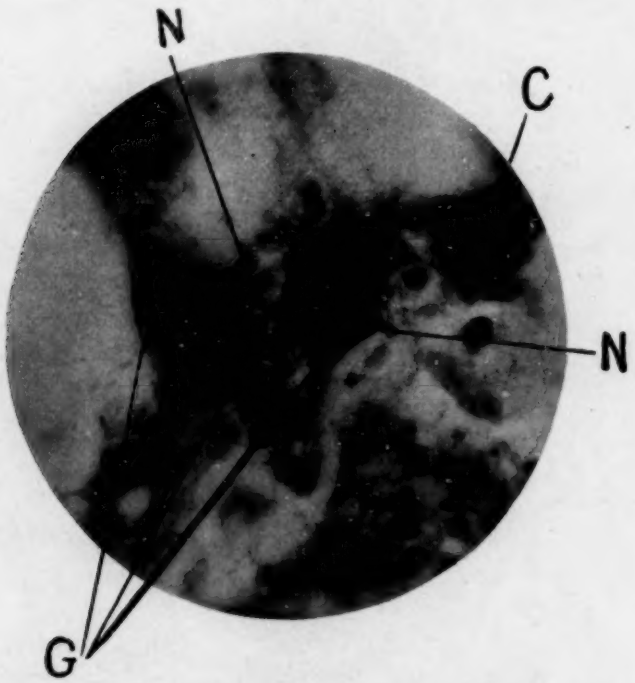

Fig. 2.—Three protoplasmic glia cells (*G*) with the nuclei (*N*, *N*) located at the very periphery. The processes of the homogeneous cell bodies are also quite distinct. At *C* is a capillary with pronounced endothelial cells and a shoe-shaped polymorphonuclear leukocyte. Alzheimer-Mann stain; ×800.

Summary of Findings.—The findings were: imbecility, loss of spatial sense, marked paralysis of the third, fourth, fifth, sixth, seventh, ninth and twelfth nerves, with partial involvement of the tenth and eleventh nerves on both sides.

The diagnosis was: imbecility, poliencephalitis superior and inferior.

Necropsy Findings.—(Permission was given to remove the brain only.)

The pia was smooth, easily detachable from the brain and shiny, except the portion over both occipitoparietal regions which showed marked hemorrhages. The convolutions were of normal size, not flattened and the sulci of usual depth. The gray matter of the midbrain and medulla appeared unusually dark, almost black, sharply contrasting with the surrounding white substance. Foci of hem-

orrhages or softening were absent; the ventricles were not enlarged and the ependyma not proliferated.

Microscopic Examination.—Various portions of the cortex, midbrain, medulla and cerebellum, including the pia-arachnoid and both third nerves, were studied in celloidin, paraffin and frozen sections. The staining methods used were toluidin blue, thionin, Alzheimer-Mann, Bielschowsky, Herxheimer and combined Alzheimer-Mann-Bielschowsky. As the main pathologic changes were expected in the midbrain and medulla, these regions will be described in somewhat greater detail.

Photomicrograph 1, showing the condition of the tegmental portion of the pons, gives a fair idea of the principal changes encountered in the midbrain

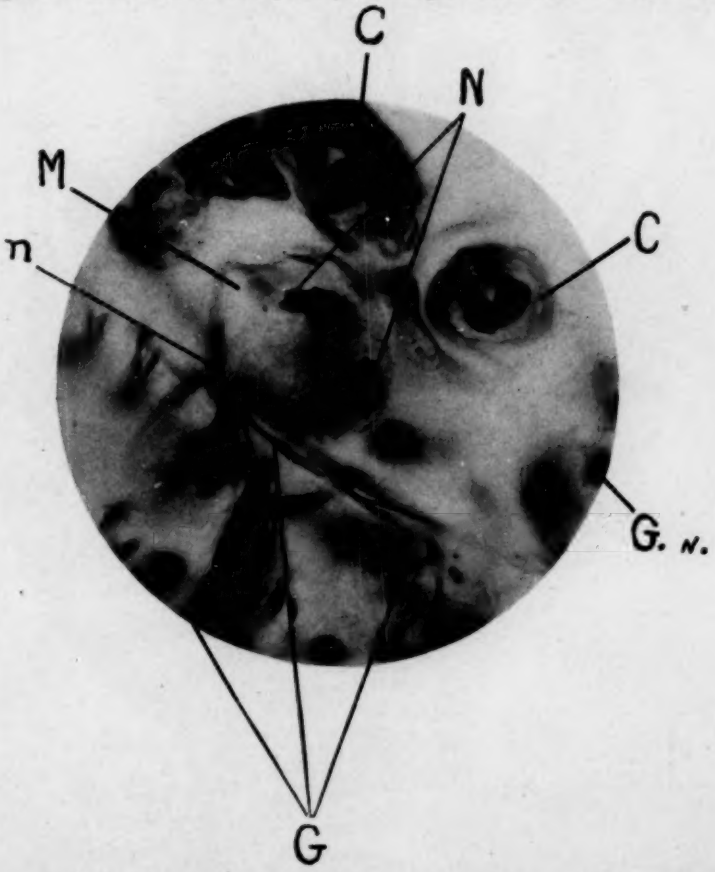

Fig. 3.—Tegmental region of the pons. *M* is a myelophage with a nucleus (*n*) and two fragments of an axon (N). *C, C* are capillaries containing four and five blood cells. The thick and other dark stained strips (*G*) are glia fibers; *G. n.* are glia nuclei. Bielschowsky stain; × 1200.

and medulla. The visual field consisted of vacuolated glia tissue with wide meshes and a great amount of hyperemic and distended vessels. The smallest capillaries were unusually prominent and engorged with blood (Figs. 2 and 3). Often there could be found newly formed capillaries in the form of elongated

adventitial and hypertrophied, brightly stained endothelial cells. Such young vessels usually were free from blood elements. The adventitial spaces of larger as well as of smaller vessels, did not show infiltration cells, such as lymphocytes, plasma cells or polyblasts. Neither could polymorphonuclear cells be found, while in some instances erythrocytes could be seen scattered freely or enclosed within various gliogenous formations. In the large ganglions, especially the lenticular nucleus, the vascular walls sometimes appeared thickened and some-

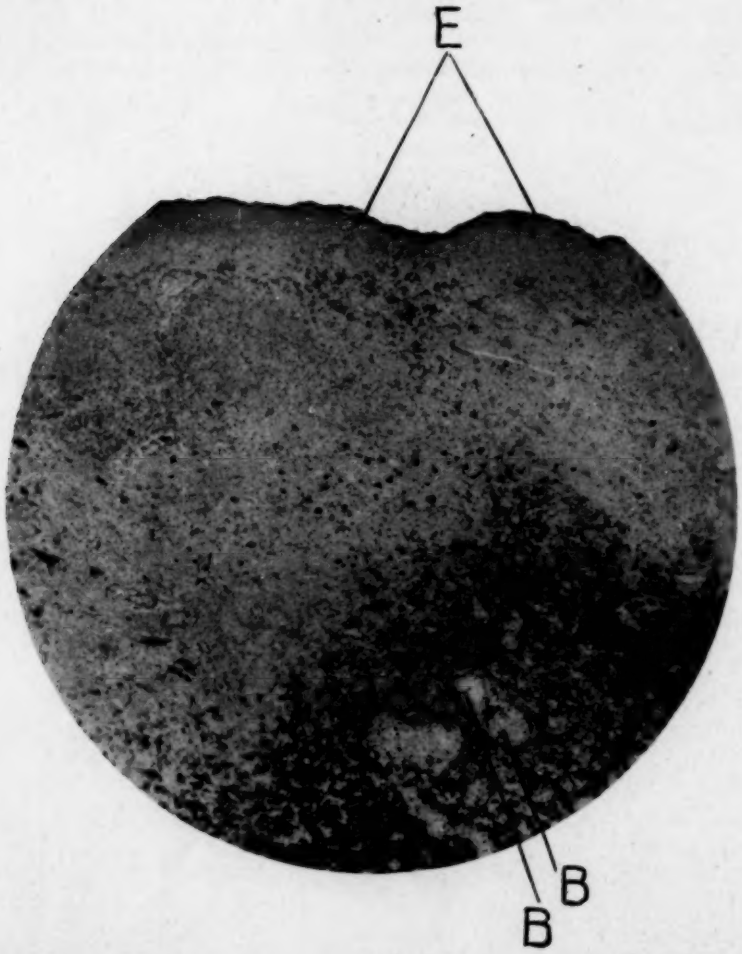

Fig. 4.—The region of the twelfth nucleus. The black masses are fat globules covering the area occupied by the twelfth nucleus and scattered as small droplets over the subependymal region. *B B* are blood vessels surrounded by fat enclosed within gitter cells; other pale areas in the darkened area are mostly vessels; at *E* is the ependyma of the fourth ventricle. Herxheimer scarlet-red-hematoxylin stain; × 60.

times showed proliferation of adventitial and endothelial elements with an abundance of brownish-green, round granules. Similar vascular changes also

could be found in the cortical layers, especially in the occipital lobe, to a lesser extent in the cerebellum, and were quite pronounced in the choroid plexus.

The characteristic feature of the vascular changes was the almost universal presence of fat within or around the vessel walls, including those of the smallest capillaries. The latter, stained with scarlet red, showed enormous quanti-

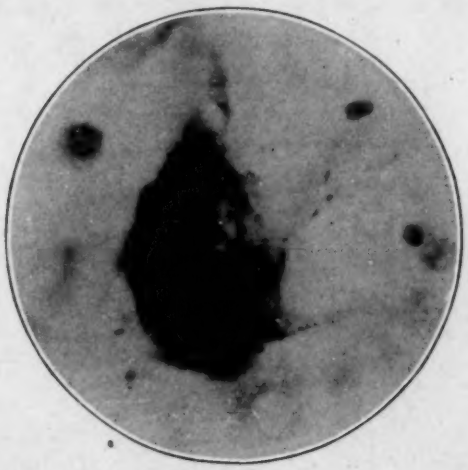

Fig. 5.—One of the types of ganglion cell changes; central chromatolysis and other changes. Toluidin-blue stain.

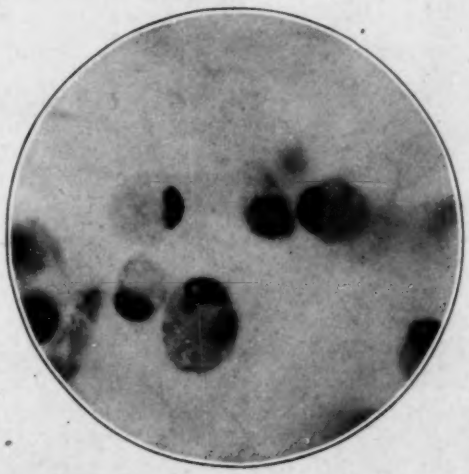

Fig. 6.—Gitter cells (γ) of one of the degenerated areas. One cell contains two nuclei. Toluidin blue stain; $\times$ 1200.

ties of fat which covered the entire visual field (Fig. 4), and made it impossible to discern any other structures except vessels and their contents.

Ganglion Cell Changes.—The ganglions sometimes appeared quite normal, even in those regions of the midbrain which exhibited marked structural

changes in the glia and blood vessels. Many nerve cells were decidedly pathologic, appearing atrophied, excavated and surrounded or invaded by a great number of glia cells. Other cells were sclerosed or homogeneous with deeply stained dendrites, dustlike chromatin and a dislocated nucleus poor in chromatin but possessing a well preserved membrane (Fig. 5). In short, chromatolysis, satellitosis and neuronophagia were the usual changes found in the ganglion cells. Stained with scarlet red, they always showed an abundance of fat drops and droplets over the entire cell body, including the nucleus.

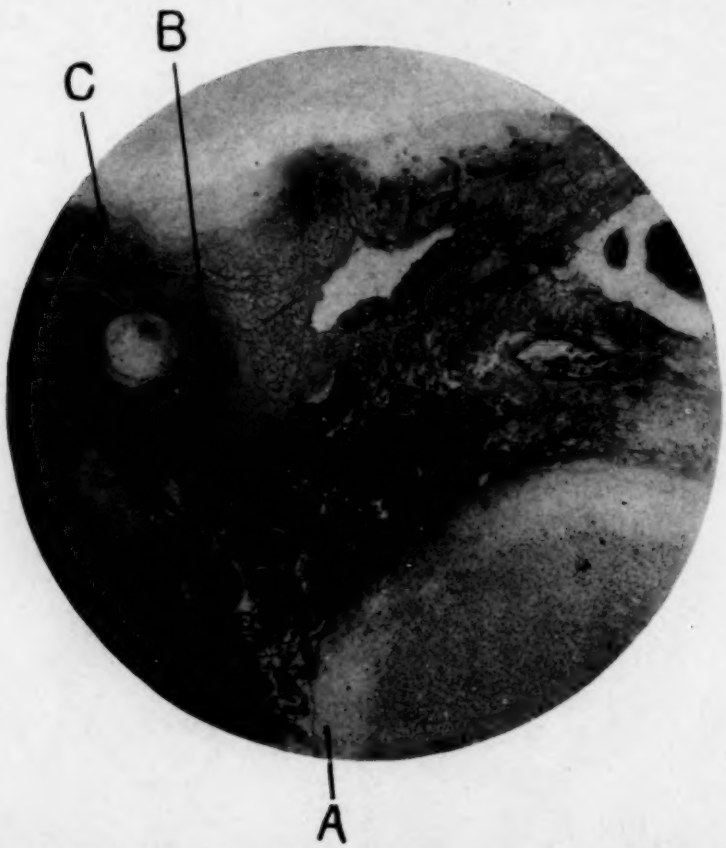

Fig. 7.—Cortex of the angular gyrus covered by a pia-arachnoid which is infiltrated with various cells and shows an abundance of blood vessels. The subpial layer *A* appears hyperplastic; *B*, hemorrhagic focus; *C*, arachnoid membrane. Toluidin-blue stain; × 16.

The foregoing cellular changes, the fat-laden ganglion cells, together with the vast accumulation of fat around the extremely numerous vessels, were especially marked around the third and fourth ventricles and the Sylvian aqueduct; that is to say, they involved the nuclei of all cranial nerves, from the third to the twelfth, including the locus coeruleus, while the gray matter of the substantia nigra, nucleus ruber and basal portion of the pons were either entirely normal or showed but mild changes.

Glia Tissue Changes.—Marked as the changes in the gray matter were, those of the neuroglia were much more striking. The wide meshes of glia, appearing under the low power as empty vacuoles, exhibited, under the high power, especially with oil immersion, a great variety of unusual structures: (1) large

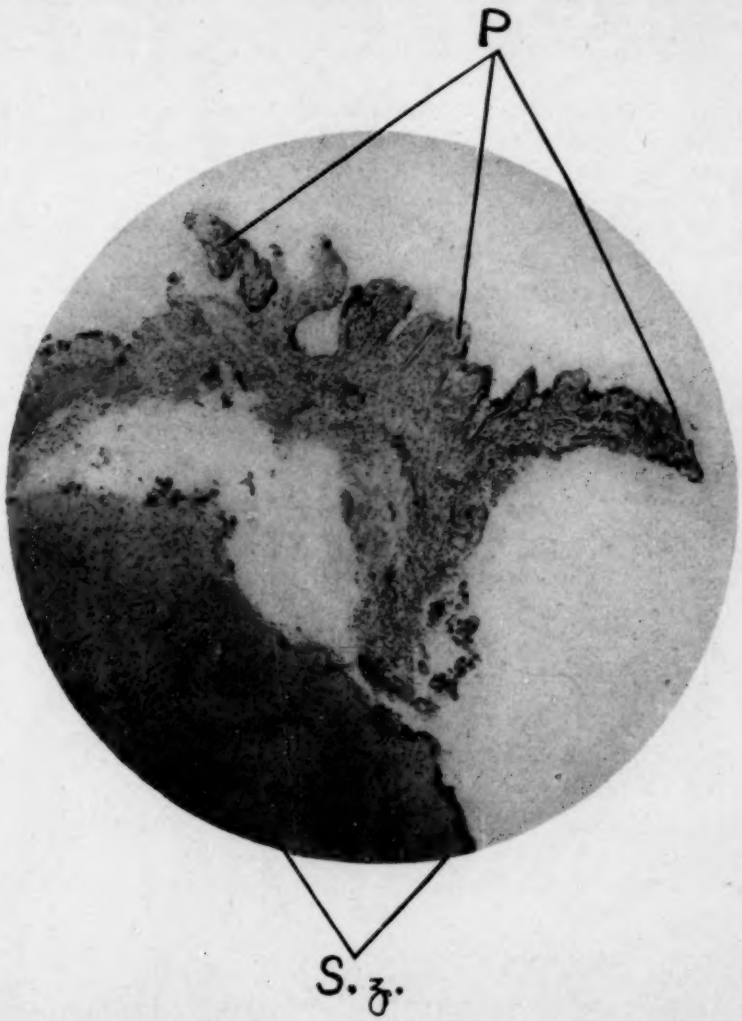

Fig. 8.—Pia-arachnoid of the motor area. The arachnoid is greatly infiltrated, mostly with gitter cells and mesothelial cells reproduced on photomicrograph 9. The arachnoid villi (pacchionian bodies) are well shown. *P* represents the pacchionian bodies (arachnoid villi); *S. z.*, the stratum zonale (subpial layer) is hyperplastic and covered by an abundance of glia nuclei. Toluidin-blue stain; × 36.

round lattice-like reticular bodies, commonly described as "gitter" cells; (2) fragments of myelin and axons; (3) red blood corpuscles; and (4) various gliogenous formations (protoplasmic glia cells, myelophages).

The gitter cells were the most numerous elements, mostly in the form of round bodies wholly made up of fine vacuoles and without processes; their nucleus was flattened and misplaced to the periphery (Fig. 5). Some cells contained more than one nucleus and were abundant around capillaries and smaller vessels. Equally numerous were gitter cells that, in addition to the great mass of small vacuoles, exhibited one large vacuole containing remnants of broken-up nerve tissue. These are so-called gitter cells—α according to Jacob's classification.

Scarlet red stained specimens frequently exhibited a third variety of gitter cells, irregular in shape and possessing a great many processes packed with fat (gitter cells—β).

Aside from these three varieties of gitter cells, there were numerous other round formations with several large vacuoles, of which some were filled with

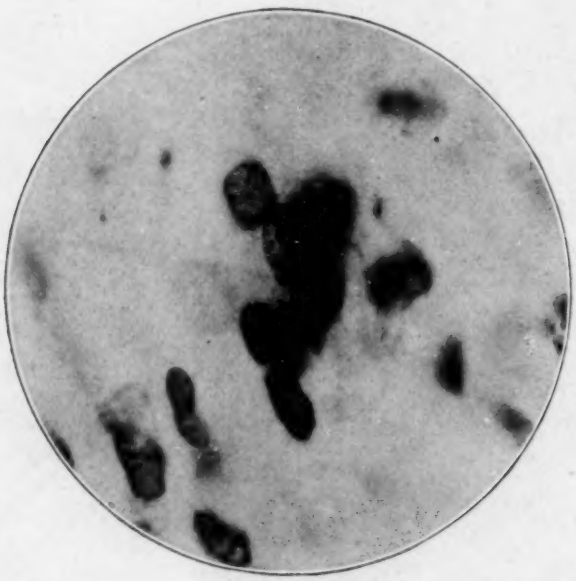

Fig. 9.—Clusters of mesothelial cells of the subarachnoid space. Toluidin-blue stain; × 1200.

red blood cells and remnants of axons and myelin, well shown in photomicrograph 3. These are myelophages (Jacob) which, like the foregoing types of gitter cells, are gliogenous formations with a specific function. For instance, the function of the myelophages is to pick up the broken-up myelin or axon and transform it into fat which is stored up in the gitter cells. It is probable that the myelophages themselves become transformed into gitter cells after their contents, myelin and axon, have been, so to speak, digested and transformed into fat. The γ-variety represents the ripe stage of gitter cells and is to be found freely scattered or around the vessels to which they are supposed to deliver the fat for final elimination.

In the large ganglions (lenticular nucleus, optic thalamus, caudate nucleus) the basal portion of the pons, cerebral and cerebellar cortices, as well as in the

subarachnoid space only the γ-variety could be encountered, the other types including myelophages, being absent.

There were in the midbrain and medulla a great number of other atypical glia cells, so-called protoplasmic glia cells. Figure 2 shows such formations, rich in protoplasm, homogeneous in appearance and well supplied with processes. The chromatin-rich nucleus was misplaced toward the periphery of the cell body as if about to desert it. The processes, in many cells, were very large and stretched for a long distance, breaking up into numerous glia fibers. Often they enveloped red cells or remnants of broken-up brain tissue, such as ganglion cells, myelin globules and even smaller capillaries. The size of the proto-

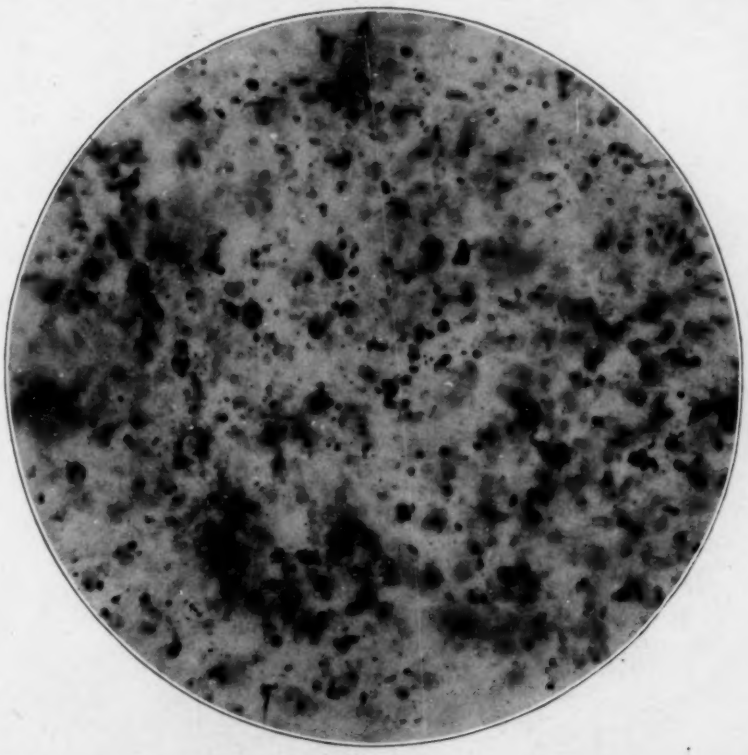

Fig. 10.—Fat globules (black dots) in the subarachnoid space. Herxheimer scarlet-red stain; × 230.

plasmic glia cells may be quite large, somewhat resembling the monster or giant glia cells of a spinal cord involved in secondary degeneration.

Finally, many glia cells were represented by mere nuclei which, however, were always increased in size and rich in chromatin. This type of glia cells predominated in the larger ganglions and basal portion of the pons and cortex. Stained with scarlet red they showed minute globules of fat.

Nerve Fibers.—These were frequently pale, poorly stainable, sometimes swollen or tumefied, and therefore irregular in shape. Frequently they were broken up into smaller fragments enveloped by glia fibers; or they formed globules of myelin (Marchi globules) enclosed within the previously described

gliogenous formations. Frequently a changed nerve fiber was accompanied by, or made up of, strips of thin, delicate, densely red fibrils (degenerated axons).

The cortical layers, which were studied from every portion of the brain and cerebellum, showed changes analogous to those of the large ganglions, namely, excessive vascularization, numerous protoplasmic glia cells filled with fat droplets and various ganglion cell changes (neuronophagia, cell sclerosis and chromatolysis). Equally well represented all over the cortex, they were especially marked in the occipital lobe, the region of cuneus and precuneus and especially the angular gyrus.

The latter was studied with particular care, in view of the history of disturbances of the spatial sense which is supposed to be located in this portion of the cortex. Photomicrograph 7 shows this part of the cortex covered with

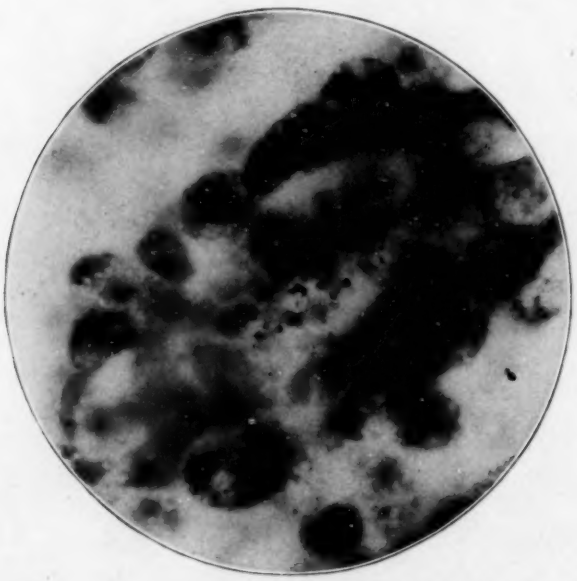

Fig. 11.—Choroid plexus. An engorged vessel surrounded by enlarged cystic epithelial cells showing granules within the cytoplasm. Toluidin-blue stain; × 800.

an enormously distended and hemorrhagic pia-arachnoid which also exhibits unusually interesting findings over the rest of the cortex, especially on the base of the brain.

The distended meshes of the pia-arachnoid were packed with a great amount of various types of cells (Figs. 7 and 8). The latter were principally mesothelial (Fig. 9) and gitter cells mixed with an abundance of red cells. There were also in evidence numerous minute granules covering the meshes, the mesothelial (around their nuclei) and partly the gitter cells. Surface frozen sections of the pia arachnoid exhibited great masses of fat (Fig. 10) enclosed within gitter cells and gathered around the capillaries. The latter were numerous, distended, showed well stained adventitial and endothelial cells but no infiltration elements, such as lymphocytes, plasma cells, etc.

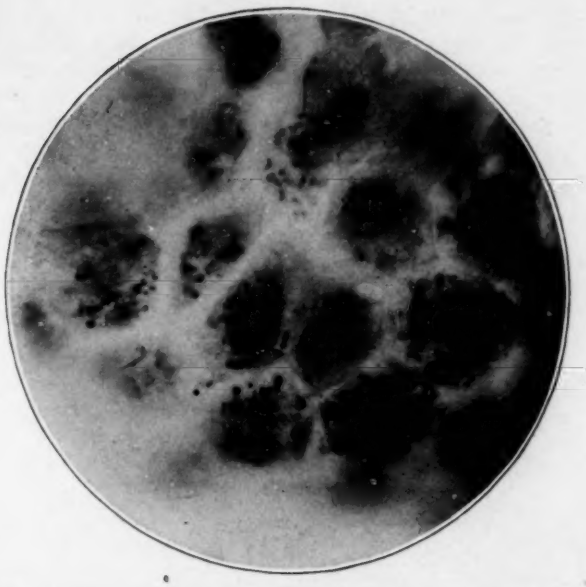

Fig. 12.—The epithelial cells of the choroid plexus packed with fat granules. Herxheimer scarlet-red; × 1200.

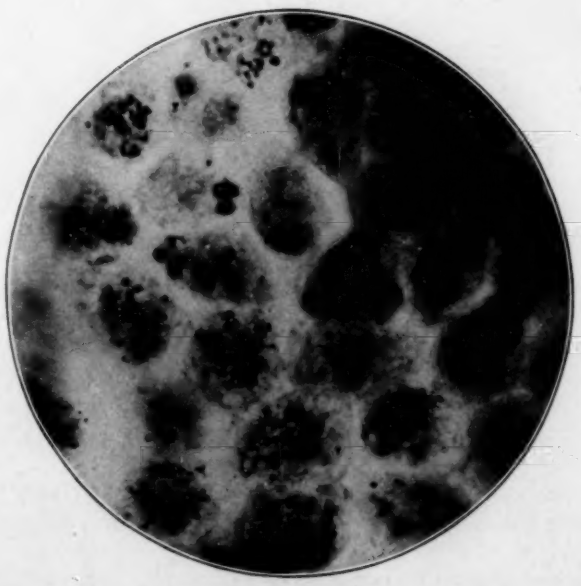

Fig. 13.—Same as photomicrograph 12. Some of the cells assumed the shape and form of gitter cells. Herxheimer scarlet-red stain; × 1200.

In some toluidin blue specimens could be seen cell bodies with pale chromatin-poor nuclei surrounded by a rim of metachromatically stained cytoplasm. They were mostly irregular in shape and numerous in the arachnoid portion of the pia. Such ill-defined elements most probably were changed mesothelial cells which, like the typical cells, could be seen all over the subarachnoid space, regardless of the localization. They were quite numerous on the base, while over the angular gyrus they were obscured by extensive hemorrhages.

Noteworthy changes were also found in the choroid plexus. The vessels were engorged, the endothelial and adventitial cells of the capillaries promi-

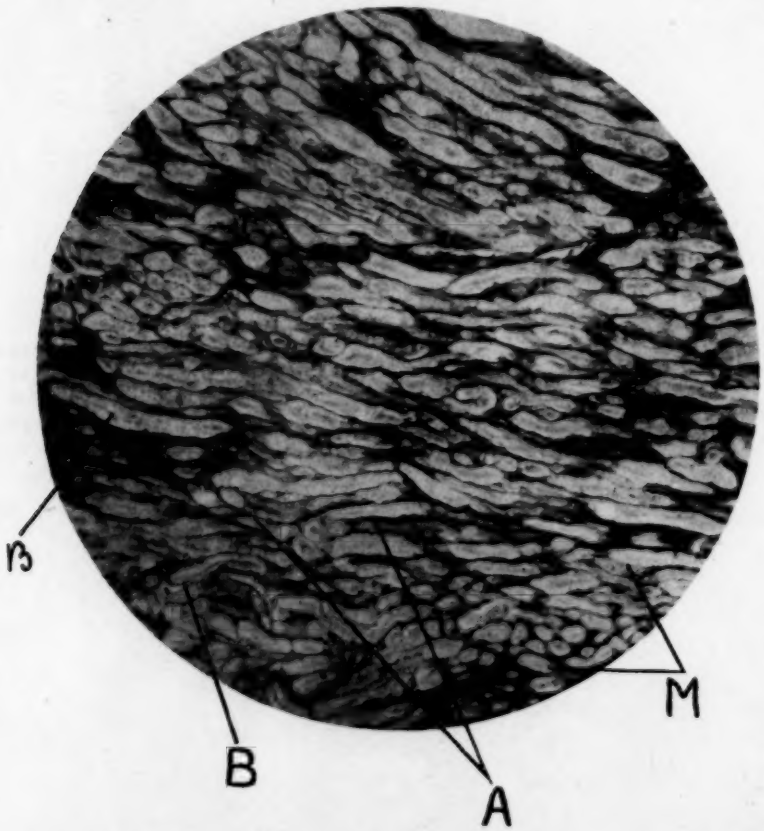

Fig. 14.—The third nerve broken up into numerous fragments; the axons can be well seen in the majority of the nerve fibers. The right lower area is filled with Marchi globules. *A* represents the Schwann membrane; *B B*, axons; *M*, Marchi globules. Alzheimer-Mann-Bielschowsky stain; × 150.

nent and the epithelial cells surrounding the vessels appeared as unusually large, expanded, reticular bodies which were well stainable and contained a peripheral nucleus (Fig. 11). Unstained specimens exhibited the usual black granules, while those stained with scarlet red showed a great number of fat droplets scattered over the cytoplasm (Figs. 12 and 13).

The third nerve, in its distal portion, was converted into numerous so-called Marchi-globules (Fig. 14), that is to say, fragments of myelin and axons, enveloped by proliferated glia tissue (Schwann cells). These changes are well represented in Figure 14 and are characteristic of secondary nerve degeneration in its early stages.

The spinal cord changes could be studied only in the upper cervical portion, which showed mild vascular and parenchymatous changes. These were even less pronounced than those of the cortex or basal ganglions.

Summary of Findings.—The findings were: widespread degenerative changes of every portion of the cerebrum and cerebellum with particular involvement of the midbrain and medulla; marked secondary proliferative changes in the glia tissue and, to a lesser extent, in the vessels; fatty infiltration of the vessels of the brain, pia-arachnoid and choroid plexus.

DISCUSSION

Quite throughout, the changes were particularly striking in the midbrain and medulla, where they exhibited a typical picture of various stages of so-called secondary degeneration, as described by A. Jacob in experimental animals. Jacob's [1] findings, discussed by me [2] at some length elsewhere, resemble the conditions to be found in amyotrophic lateral sclerosis, subacute combined cord degeneration, progressive bulbar paralysis and, to a large extent, in tabes and multiple sclerosis. The case under discussion differs from the foregoing by the principal, though not exclusive, involvement of the midbrain and medulla oblongata. In this respect it is almost analogous to another mesencephalic lesion—lethargic or epidemic encephalitis. The latter, however, is a purely inflammatory process, due to some infection, while the case under discussion is not inflammatory or infectious. It is most probably caused by some organic toxin, as is evidently the case in amyotrophic lateral sclerosis, subacute combined cord degeneration and similar conditions.

The term poliencephalitis superior hemorrhagica applied to certain cases, including the present one, is therefore a misnomer, as already pointed out by Schroeder,[3] among others. First of all, hemorrhages are not essential. They may be entirely absent (Oppenheim and Cassirer [4]), or, as in this case, be so small that only careful microscopic search reveals them. It is therefore altogether illogical to look on these microscopic hemorrhages as the cause of the widespread degenerative changes

1. Jacob, A.: Ueber die feinere Histologie der secundären Faserdegeneration in der weissen Substanz des Rückenmarks, Nissl-Alzheimer's Arb. **5**:1, 1912.

2. Hassin, G. B.: Histopathological Changes in a Case of Amyotrophic Lateral Sclerosis, Med. Rec. **91**:228 (Feb. 10) 1917.

3. Schroeder, Paul: Zur Lehre von der akuten hämorrhagischen Poliencephalitis superior (Wernicke), Nissl-Alsheimer's Arb. **2**:145, 1908.

4. Oppenheim, H., and Cassirer, R.: Die Encephalitis, in Nothnagel's Specielle Pathologie und Therapie, Wien. **9**: Pt. 2, 39, 1909.

and the marked progressive glia phenomena, an opinion expressed by Schroeder. The cause as stated is probably some organic toxin or catabolic phenomena which seems to possess the peculiar ability to affect certain portions of the central nervous system leaving others intact. For instance, the pure motor elements may alone be affected and give rise to the clinical syndromes of amyotrophic lateral sclerosis, bulbar paralysis or progressive muscular atrophy; or the white substance, principally of the spinal cord, may be exclusively involved, leaving the motor and sensory elements undamaged and thus produce a condition of subacute combined cord degeneration. The action of the problematical (catabolic) toxins is thus selective causing a great variety of morbid conditions which, though pathologically similar, are dissimilar in localization.

The histologic studies of this case bring forth some other interesting facts. The presence of fat granule bodies (gitter cells) in the subarachnoid space and the choroid plexus, as well as in the perivascular spaces, all over the brain stem and the cerebellum can be explained only by assuming that they reached these structures, or had been transported to them, from the degenerated areas of the midbrain. In other words, the great masses of fat were gradually drained from the midbrain regions by way of perivascular spaces toward the subarachnoid space.

The latter thus may be looked on as the receptacle of the waste products discharged by the brain tissues. Some of these waste products are lipoid substances, some probably are represented by the numerous granules in the subarachnoid space. The proliferation of the mesothelial cells and the abundance of other, mostly ill-defined, elements is probably a reaction against the invading waste products, just as they react against invasion by some particular matter (Essick,[5] Weed[6]).

When these products reach the subarachnoid space they are removed thence by the cerebrospinal fluid. If the latter is to be considered as the product of the choroid plexus, then the function of the cerebrospinal fluid is to wash off the waste products that come from the brain to the subarachnoid space.

The choroid plexus, however, exhibited an abundance of fat in the epithelial cells and its vessels. The fat could come only from the spinal fluid which thus gets rid, with the help of the choroid plexus, of some of its contents—in this case of the lipoids.

One gains an impression that the spinal fluid is entirely made up of the tissue fluids of the brain and is being continually discharged by way

5. Essick, C.: Formation of Macrophages by the Cells Lining the Subarachnoid Cavity in Response to the Stimulus of Particulate Matter, Publication 272 of the Carnegie Institution in Washington, pp. 377 and 388.

6. Weed, L. H.: The Cells of the Arachnoid, Bull. Johns Hopkins Hosp. **31**:343 (Oct.) 1920.

of the perivascular spaces of the cerebral vessels into the subarachnoid space. Weed [7] admits such a possibility though he believes that only small amounts of cerebrospinal fluid originate in this manner. Yet, coming as it does from such a voluminous and active organ as the brain, the cerebral fluid necessarily must be abundant and rich in proteins and various waste products. Before being absorbed by the arachnoid villi and other channels, the fluid becomes cleared, as it were, by the choroid plexus and thus rendered more passable.

Further pathologic studies of cases like the one recorded will, to a great extent, facilitate the solving of these highly difficult and interesting problems. The facts brought forth by histopathologic studies of the subarachnoid space and the choroid plexus may show that the function of the cerebrospinal fluid and the choroid plexus is altogether different from the one generally accepted.

CONCLUSION

1. Poliencephalitis superior (hemorrhagica) of Wernicke is not an inflammatory, but a partial manifestation of a general degenerative process of the central nervous system.

2. It is analogous to other degenerative processes, such as amyotrophic lateral sclerosis, subacute combined cord degeneration, progressive bulbar paralysis and tabes dorsalis, from which it differs by the localization of the degenerative phenomena.

3. It is essentially, though not exclusively, a mesencephalic lesion, resembling in its localization epidemic encephalitis which, however, is not a degenerative but an inflammatory process.

4. The subarachnoid space is a receptacle of the tissue fluids which carry away the waste products of the brain.

5. The function of the choroid plexus is probably to pick up from the cerebrospinal fluid harmful or other products and to render them, as well as the fluid, more absorbable.

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7. Weed, L. H.: Studies on Cerebrospinal Fluid, Article IV: The Dual Source of Cerebrospinal Fluid, Jour. Med. Research **31**:93, 1914.

THE MONGOLIAN IDIOT

A PRELIMINARY NOTE ON THE SELLA TURCICA FINDINGS

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NEW YORK

In the past year it has been my great privilege to examine a number of Mongolian defectives at Waverley, Mass., at the invitation of Dr. Walter E. Fernald. The case reports, complete clinical data, biochemical findings and the result of treatment on an endocrinologic basis will be the substance of a later communication by Dr. Fernald and myself. I desire to make at this time a preliminary publication of the findings of the roentgenologic examination of many of the skulls for the reason that it may stimulate the making of such examinations in many centers with possible corroboration of my findings. Furthermore, if necropsy material is available, a special effort might be made, on the basis of the sellar changes shown in the radiographs, to determine the pathology of the pituitary body.

In twenty-three out of twenty-four nonselected cases of Mongolian idiocy, including six of my own, the radiograph of the skull showed a peculiar change from the normal in the anterior portion of the fossa pituitaria. This change consisted in an excavation under the anterior clinoid processes and presumably under the olivary process and optic groove, and the excavation communicated directly with the anterior portion of the fossa itself. There were varying degrees of this excavation, as seen in the accompanying illustrations. From our knowledge, more or less exact, of the influence of the anterior lobe of the pituitary on growth and genital development, this roentgen-ray finding is of considerable interest. Especially is this true when we remember that among the clinical signs of Mongolian idiots we invariably have the combination of subnormal and disproportionate body growth coupled with lack of genital development. Thus in boys, undescended testicles are quite frequent and in girls abnormalities of the genitals with tardy menstrual flow or even complete amenorrhea are the rule. The intimate relationship which in early life exists between the anterior hypophysial lobe and the pharyngeal glandular elements is also strikingly coincidental with the extreme pharyngeal mucous secretion seen in Mongolian idiots. Furthermore, with such an excavation, involving at times the optic groove, eye symptoms should be of frequent occurrence. These

ocular manifestations have been the subject of much discussion among ophthalmologists and a rather complete résumé of their findings was published some years ago by Charles A. Oliver.[1] Among the findings are:

"The optic discs in a number of cases are unequally grayed, especially in the deeper layers and to the temporal sides; the substance of the disc in the great majority of cases, especially where the retinal and choroidal disturbance was not pronounced, is apparently edematous; the retina surrounding the optic nerve head is edematous and swollen in many of the cases; examination for visual field disturbances could never be properly carried out through lack of cooperation."

Theoretically, therefore, disturbance of the anterior portion of the pituitary body might readily produce many of the symptoms shown clinically by Mongolian idiots. It is therefore suggested that every necropsy examination in cases of Mongolian idiocy should include a careful examination of the pituitary gland, notably in its anterior portion. Should such an examination eventuate in a corroboration of antemortem findings, perhaps a rational treatment might be forthcoming for these cases. Indeed, even on the theoretical basis of anterior lobe disturbance, the writer has inaugurated a therapy in several of his cases which has had some degree of success thus far. In one of his Mongolian idiots the testicles have descended since treatment was begun, and there seems to be a measure of mental improvement likewise. This treatment includes the hypodermatic injections of anterior lobe extract (antuitrin) combined with whole gland feeding and thyroid administration in small doses.

It is important to observe, nevertheless, that excavation under the anterior clinoids and optic groove is seen occasionally in persons who have not Mongolian characteristics, but who have isolated symptoms and signs due to anterior lobe disturbance, namely, growth and genital developmental abnormalities.

Speculation as to the character of the contents of this excavation would at the present be idle. Suffice it to say, however, that on theoretical grounds it should be tissue foreign to the normal anterior lobe type and possibly interfering with whatever of normal structure may remain in the region.

There is included in the series of photographs accompanying this communication one of a Chinese girl, 13 years of age, who is normal in all ways, in order that a comparison may be made of her sella turcica

1. Oliver, C. A.: Med. Rec., Oct. 3, 1891.

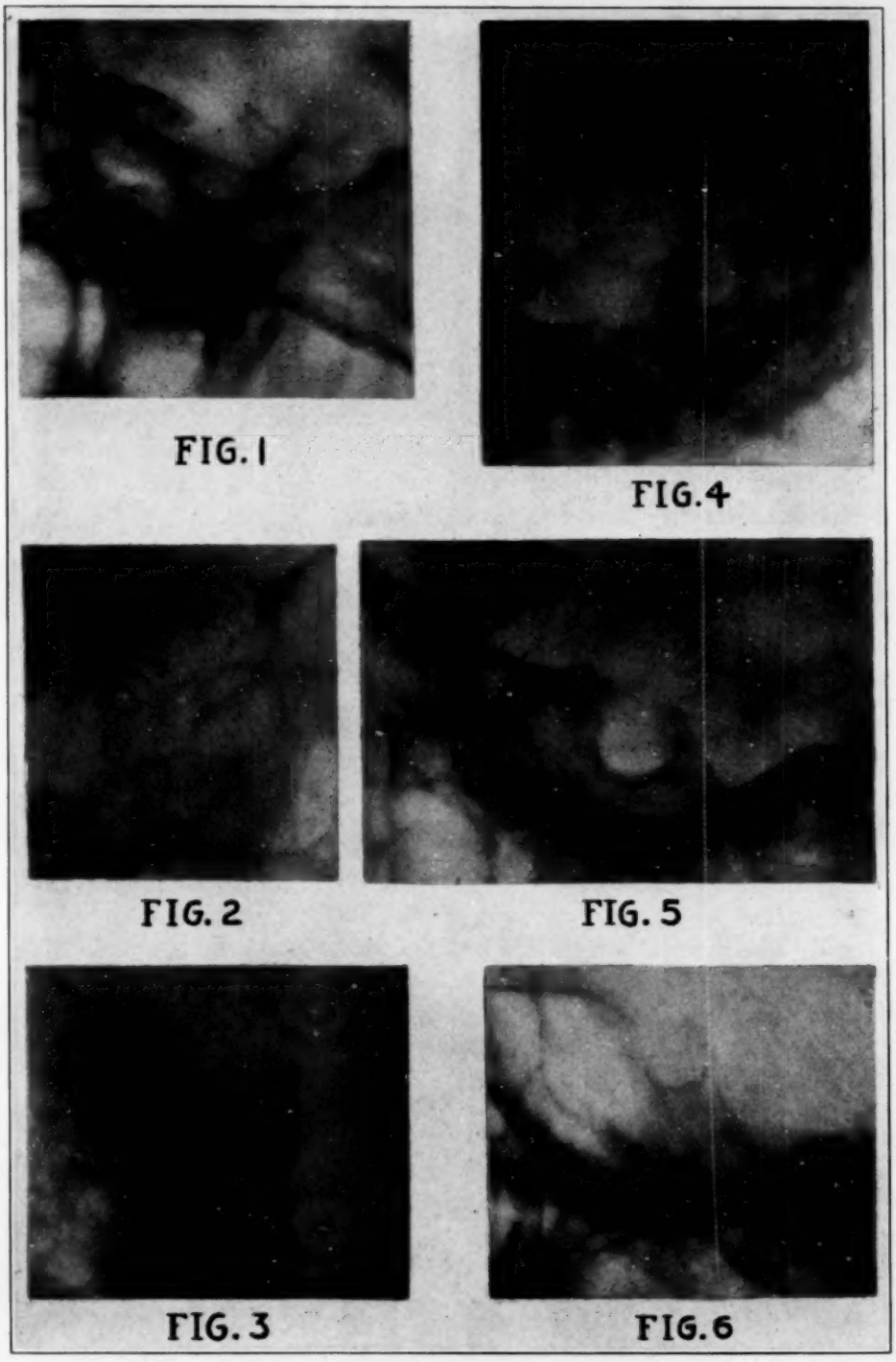

Fig. 1 (T. J., girl, Waverley).—The sella turcica is rather generally enlarged with a marked excavation anteriorly opening directly into the general pituitary fossa.

Fig. 2 (S. H., girl, author's patient).—Anterior excavation prominent and even somewhat larger than the pituitary fossa itself.

Fig. 3 (T. S., boy, Waverley).—Similar to Figure 2.

Fig. 4 (L. H., girl, author's patient).—Anterior excavation rather different from foregoing in that while it is fairly long, its width is less.

Fig. 5 (E. M., girl, Waverley).—Similar to Figure 4, with a narrow and rather short cavity anteriorly.

Fig. 6 (Chinese girls).—Normal sella turcica. Kindness of Percy Ashley.

with that of the Mongolian idiot. It will be seen that in her skull the pituitary fossa seems to be normal in all particulars, however different the other skull landmarks may appear to be.

From time to time observers have been adding additional findings in their reports on Mongolian idiocy, but these findings have been practically all in the symptomatologic domain. The present report of

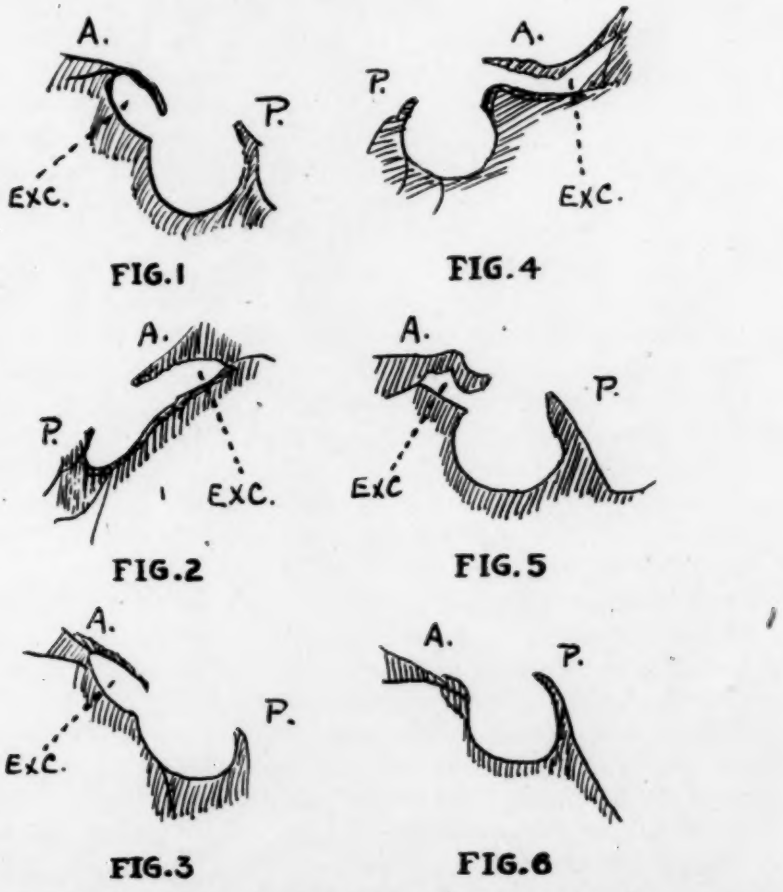

Fig. 7.—Diagrams illustrating sella photographs. A, anterior clinoid process, P, posterior clinoid process; Exc., excavation.

the sellar changes, if substantiated by the necropsy examination, is of quite a different nature, for it furnishes at once a basis for the structural changes of the Mongolian idiot and for the greater part of his symptomatology, and hence must be regarded as one of the fundamentals of the situation. Furthermore, it offers an angle of attack in therapy.

SYPHILIS OF THE EIGHTH NERVE*

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In view of the fact that there is no affection of any cranial or spinal nerve that is more characteristic or more easily recognizable, and scarcely any other, unless it be invasion of the optic nerves, that is followed by more lamentable results, it is rather remarkable that syphilitic disease of the eighth nerve had apparently attracted but little notice among American and English neurologists until comparatively recent years. This was so in spite of the fact that Jonathan Hutchinson had called attention to it, and had even made it one of the triad of symptoms in hereditary syphilis. He had also referred to absolute deafness coming on rapidly in secondary syphilis, usually bilateral; but he had given no systematic description of the symptoms that distinguish the affection of the cochlear nerve from that of the vestibular. Gowers has an inadequate reference to the subject; he speaks of primary degeneration of the eighth nerve in locomotor ataxia, but he has little to say about acute syphilis of the acoustic nerve. The older American textbooks have only passing allusions to the subject. Mills gives a more complete, but condensed, account. He refers to the theory of Althaus that the vestibular suffers more than the cochlear nerve in tabes, due to a degenerative process, causing vertigo.

It is to the otologists that we must look for more complete work, because the subject is generally assigned to them, and there is a large bibliography. West, in Power and Murphy's "System," gives a good description of syphilis of the internal ear and auditory nerve. He seems to think that the early sudden cases are due to labyrinthitis, and the late tertiary cases to meningitis and neuritis. He gives a long bibliography. Rosenstein, in 1905, studied the changes in the auditory nerve, and found basilar gummatous meningitis. In Hazen's recent work on "Syphilis" the chapter on the ear is written by Dabney, who reviews the subject and gives many references, mostly to the otologists who have written on it. Fournier, in his work on "Syphilis," has a short chapter written by Hermet, who speaks of the rapidity and incurability of syphilis of the eighth nerve, some cases occurring as early as the fifth and sixth months after the primary infection, sometimes accompanied with paralysis of the seventh nerve. The best recent review of the subject in English, of which I have knowledge, is by Dr. G. W. Mackenzie of Philadelphia, in a paper on "Syphilis of the Inner Ear and

* From the wards and laboratory of the Philadelphia General Hospital.

Eighth Nerve." I must leave to the otologists their own field, for I am writing as a neurologist and largely, but not entirely, from a clinical standpoint.

My attention has been called anew to this subject by the recent occurence of early and striking cases in hospital practice. This may be due in part to the intensive study of syphilis of the nervous system which has followed upon our knowledge of the spirochete and our advanced laboratory methods. We have come to know how early the spirochete may invade the central nervous system, and we are naturally on the lookout for any and all manifestations of it. Cases of deafness, tinnitus and vertigo, which formerly would probably have been promptly sent to the otologist, are now retained by the neurologist and scrutinized most closely. This is proper and necessary, because few, if any, of these cases are strictly otologic. They are primarily nervous cases; the lesion is in the nervous system; and the involvement of the eighth nerve is nearly always associated with other well marked symptoms of nervous syphilis.

CHARACTERISTICS OF SYPHILIS OF THE EIGHTH NERVE

There are certain characteristics of syphilis of the eighth nerve which the neurologist should bear in mind, particularly as he is quite as likely as the otologist or syphilologist to see these cases in their earliest stage.

First, the onset may occur early in the secondary stage, sometimes very early; and even before the secondary stage, if we are to believe Pollitzer, who claimed that he saw a case seven days after infection. Randall saw a case in which deafness occurred four weeks after an infected needle-wound of the finger. Other writers speak of its early appearance, and this has been true in my own observation, the disorder showing itself in a few months.

Second, suddenness of onset and rapidity of course are sometimes striking. The patient may become deaf in a few days. Hermet spoke of "surdité foudroyant." Dabney says that "sudden loss of hearing, generally with tinnitus, no pain, no evidence of middle-ear disease, in a young adult otherwise healthy, should be regarded as almost certainly indicative of syphilitic disease of the eighth nerve or labyrinth." This tinnitus is usually marked and most distressing, even keeping the patient awake at night. It is, of course, evidence of involvement of the cochlear nerve.

Third, the disease is usually bilateral, seldom unilateral, as Hutchinson pointed out.

Fourth, there may be a cranial polyneuritis, the seventh nerve especially being paralyzed with the eighth. Sometimes the second, third, and fifth, one or all, are involved. Nonne and others have called

attention to this fact as evidence that the affection is primarily a basilar meningitis, not a labyrinthitis as some of the older otologists taught. As evidence that we are dealing with a basilar syphilitic meningitis is the fact that severe headache, with high lymphocytosis of the spinal fluid, may precede or accompany the neuritis. But this apparently is not so in all cases.

Fifth, the two divisions of the eighth nerve may not be equally involved. The cochlear in one case, the vestibular in another, may be the more affected. This is in accord with the well-known selective action of syphilis. The vestibular nerve should always be tested by the Bárány methods.

Finally, the disease may be incurable, causing complete deafness in a short time.

THE COCHLEAR AND VESTIBULAR NERVES, THE SO-CALLED ROOTS OF THE EIGHTH NERVE

It is to be borne in mind that the eighth nerve, although usually described by anatomists as one nerve with two branches, is really two distinct nerves, each with its own ganglion of origin, its own nerve trunk, and its own separate and distinct course and distribution in the central nervous system. For a part of their course these two nerve-trunks are bound together (in the internal auditory meatus) so that they appear as one nerve, but before entering the brain-stem they separate, and form the so-called roots of the eighth nerve—but they are not roots in any true sense, because, like all sensory neurons, these have their ganglions of origin outside of the central nervous system. The cochlear nerve arises in the ganglion spirale, or ganglion of Corti, within the labyrinth; the vestibular, in the ganglion of Scarpa, which is located within the internal auditory meatus. The fact that these two so-called roots are distinct before entering the brain-stem, explains how they can be separately affected in syphilitic meningitis. The fact that the eighth nerve has no neurilemma (hence called the portio mollis) may explain its vulnerability to the spirochete, and the extreme rapidity of the destructive action.

Disease of the cochlear nerve causes deafness of various grades, which is to be determined by tests made by the otologists. It has been claimed by some that abolition of the very high and very low notes, with preservation of the intermediate notes, hence allowing the hearing of a conversational tone, is quite characteristic. This was present in one of my cases; but Mackenzie denies that it is common, and says that various notes are abolished in various cases. Appropriate tests are likely to show impairment of bone conduction. It is necessary to exclude disease of the middle ear.

The vestibular nerve presides over equilibration, and its central connections are with nuclei in the brain-stem, from which by a second relay of neurons its ultimate connections are made, especially with the cerebellum and the nuclei in the midbrain. Vertigo and nystagmus, which are normal reactions on turning and douching, are variously affected, as can usually be demonstrated by the Bárány tests. Clinically, these patients may have various forms or degrees of vertigo, but in my observation staggering gait is not common.

I do not attempt here to describe tests and reactions in detail, for this province belongs to the otologist, whose cooperation is always essential in a proper study of these cases. I believe, however, it is necessary to be on guard against hasty conclusions as to the localizing value of the Bárány tests. In my observation these tests in these cases are mainly valuable in showing alteration or complete abolition of the normal vertigo and nystagmus on turning, the inference being that the nerve trunk is implicated.

It is not to be denied that there may be a true syphilitic labyrinthitis. Some otologists seem to have held that this was the common pathology, especially in the early acute cases. It may be so also in the hereditary cases, in which the bone also may be diseased. The distinction between disease of the labyrinth and disease of the nerve is, I understand, not an easy one, and it is a problem for the ear specialist rather than for the neurologist; but there is little doubt, for reasons which I have already given, that the meninges and nerve trunk are the seats of the disease in many cases, if not in the majority.

The following case may serve as an example of the fulminating type of this disease:

REPORT OF CASES

Case 1.—A negro, 30 years old, had been treated in the venereal wards of the Philadelphia General Hospital in June, 1917, for a chancre and secondary eruption. At this time he had received one dose of arsphenamin. He had later a syphilitic iritis, but there is no record of any nervous symptoms at this time. He left the hospital and returned to his work, but about two months later he was taken with a severe frontal headache, which forced him to return to the hospital. The headache was the only complaint. The right pupil was rigid, the result of the iritis; the left was sluggish. The cranial nerves were normal. The knee and Achilles' reflexes were much diminished, but there was no ataxia or swaying. As a routine measure the hearing was tested; no deafness was observed. Both the blood and cerebrospinal fluid were strongly positive, and there was a very high lymphocytosis. He was given mercurial inunctions and several doses of arsphenamin. In October the report from the ear clinic stated that hearing was normal; late in November, however, the hearing was slightly impaired, but the drum membranes were normal. Under treatment the headache entirely disappeared, and the blood became negative; the spinal fluid remained positive, though the cell count was much reduced. Toward March the man eloped from the hospital and was gone several weeks, when he returned. In

that short interval he had become very deaf, to such a degree that it was difficult to speak with him. He complained much of loud tinnitus. The report from the ear clinic stated that in the right ear the involvement seemed to be confined to the labyrinth, but in the left ear the indications were that the lesion was in the course of the nerve. Bárány tests were not made, but the patient had no vertigo or staggering. Active treatment did not relieve the deafness, which indeed increased until in a short time it was practically complete. There was a smoothing out of the face and brow on the left side, due to slight paresis of the seventh nerve.

The Bárány tests may give interesting results in some of these cases, as the following instance shows. It also shows bilateral seventh nerve palsy, which in itself is a rarity.

Case 2.—The patient, a colored man, aged 23, had a primary sore in September, 1918. He was admitted in October to the venereal wards of the Philadelphia General Hospital, where he was given two doses of arsphenamin; he left the hospital against advice on December 1. Three weeks later (or about three months after the appearance of the chancre) he began to have severe and persistent headache, accompanied with vertigo and tinnitus in both ears. He was readmitted to the hospital in January, when it was observed that he had paralysis of both facial nerves, more marked on the left. Other cranial nerves were not involved (with the exception to be noted) nor was there any impairment of the spinal cord, but the Achilles' reflex on the left side was abolished. The pupillary responses were normal; there was no optic neuritis. The Wassermann tests of the blood and spinal fluid were positive, and the cell count was as high as 780. He received six doses of arsphenamin up to March, 1919. The positive but shortened Rinne's test, with loss of intermediate notes, pointed strongly to involvement of the nervous mechanism; but the patient heard ordinary conversation. Dr. Lewis Fisher reported total absence of response after douching and turning, which would indicate a lesion of the eighth nerve (vestibular division); but a definite presence of vertigo after turning, as well as a preservation of a fair amount of hearing on both sides, showed that the eighth nerves were not involved in their entirety. Dr. Fisher suggested a bilateral lesion of the brain-stem on the mesial aspect of Deiters' nucleus, thus allowing the escape of the fibers for vertigo and most of the auditory fibers; but the involvement of both seventh nerves was against this view, for it clearly indicated a peripheral lesion of the so-called roots of the eighth nerves, especially the vestibular, along with the seventh nerves, in a syphilitic meningitis. As already said, it is quite conceivable that these two roots may not be equally involved in a meningitis. It is noteworthy that this patient's seventh and eighth nerve involvement showed itself three months after the primary lesion, and after he had received two doses of the arsenical drug.

The following case is of especial importance because it is the only case in the series in which it was possible to examine the eighth nerve under the microscope. It is also of much clinical interest. As in the two preceding cases, the patient was under observation from the time of the initial lesion.

Case 3.—The patient, a white man, aged 48, was admitted to the venereal wards in September, 1918, with a primary sore. He received five doses of arsphenamin. In March, 1919 (six months after the primary sore), he began

to have a staggering gait, which caused him to fall to the left. Trouble with hearing had commenced earlier in the right ear, in which he had become quite deaf; later the left ear failed. There was loud tinnitus. The blood Wassermann reaction was reported negative, but the spinal fluid was ++++. The cell count was 910. There was complete abolition of the left Achilles' jerk, just as in the other case (an odd coincidence). A low-grade double optic neuritis was present, but the pupils acted normally. The frontalis muscle on the left was smoothed out. All the other cranial nerves were normal. Dr. Fisher found that the right labyrinth was completely, the left partly, involved. There were no reactions to turning or douching. The case seemed to be a clear one of peripheral involvement, that is, of the labyrinths and eighth nerves in both divisions. The earliest symptoms appeared in less than six months after the primary sore. This man was taken with acute appendicitis and died. Under the microscope the eighth nerves were seen to have been affected, as shown in the following report.

Neuropathologic Report (by Dr. N. W. Winkleman).—Pathologic study was made of sections from representative areas of the cerebrum, cerebellum, pons, medulla, spinal cord and the eighth nerves. The stains used in this study were: a hematoxylin-eosin, Weigert's myelin sheath stain, Mallory's phosphotungstic-acid-hematoxylin and Alzheimer-Mann.

The cerebrum and cerebellum are normal, especially in so far as syphilis is concerned. The pons is negative. In the medulla there is seen a round cell infiltration (lymphocytes) in the nucleus vestibularis lateralis (Deiters') with fatty degeneration of the nerve cells of this nucleus on the right side. The membranes are normal. The spinal cord is negative.

Both eighth nerves show some swelling and tortuosity of the axis cylinders, as seen in longitudinal section, with some dropping out of axis cylinders as seen in cross section. There are present within the nerves large, round, vesicular, pale staining nuclei with irregular, indefinite, pale, acid-staining cytoplasm—in all resembling glia cells, and in places fibers are seen coming off the cytoplasm. Besides these there is present another type of cell: a long, narrow, heavier staining nucleus with a definite outline to its rather meager cytoplasm—a so-called sheath cell. At certain places within the nerves is seen a slight but definite lymphocytic infiltration. Amyloid bodies are present to excess. Around some of the vessels within the nerves are a few plasma cells with many lymphocytes. The sheath of the nerves shows a very definite though not very heavy infiltration with lymphocytes.

Diagnosis: Syphilis of both eighth nerves with involvement of Deiters' nucleus on the right.

The following case, which occurred in the service of Dr. Charles S. Potts, showed improvement under treatment with neo-arsphenamin (five doses) and mercurial inunctions. It was a late tertiary case.

CASE 4.—A white man, aged 56, began, one month before admission to the hospital, to have deafness, with vertigo and tinnitus in both ears. He also had impaired sight in the right eye and slight paresis of the right seventh nerve. There was a history of a primary sore fourteen years before. The left pupil was sluggish and irregular, the right inactive to light. Sight was much impaired. The right optic disk was much congested, with blurred margins. The Bárány tests revealed horizontal nystagmus to the left, four seconds; to

the right, four seconds. The tests of the blood and spinal fluid were positive on two examinations, but the cell count was low. Improvement was rapid under treatment. There was return of function of the vestibular, as well as of the cochlear, nerve. When discharged, the man could hear and see fairly well. It is noteworthy that this was a tertiary case of long standing, and that there was no lymphocytosis. This probably puts it in a separate class from the acute cases, already described, coming on in the early secondary stage, with headache and high lymphocytosis, indicating a rapid and acute involvement of the meninges.

Case 5.—Another patient in Dr. Potts' wards was a negro, aged 28, who had become deaf rapidly, and who also had paralysis of the right seventh nerve. The pupils were irregular and sluggish. The clinical notes are not very full, as it was almost impossible to communicate with the patient, and he soon began to show mental symptoms, for which he was transferred to the department for the insane. No history of a primary sore could be elicited; the laboratory reports for blood and spinal fluid were negative, but there was a high lymphocytosis. He may have had a syphilitic psychosis. The onset of rapid and complete deafness in a young adult, with tinnitus, without obvious cause, and without disease of the middle ear is, as Dabney pointed out, significant of syphilis; and I may add that this is especially true if there is also a paralysis of the seventh nerve.

Case 6.—This patient's condition was of five years' duration. He was a white man, 31 years old. He had bilateral seventh and bilateral eighth nerve paralysis and anesthesia on the right side of his face. The blood reaction had been reported several times as strongly positive. He had come and gone to and from the hospital four or five times; consequently the course of symptoms had not been accurately traced. His condition was evidently incurable, and illustrated the lamentable fate of a patient with this syphilitic syndrome. He was almost totally deaf, with paralysis and contractures of both sides of his face.

EFFECTS OF ARSPHENAMIN ON SYPHILIS OF THE EIGHTH NERVE

In discussing this affection, the subject of neurorecidivus, or the spinal fluid changes in neurosyphilis caused by a provocative dose of arsphenamin, inevitably occurs to the mind. The first three of the foregoing series of cases suggest this possibility. Another thing to be considered is the possible injurious action of the drug itself directly on the nerves. This charge has been made against this arsenical preparation not only in the case of the eighth nerves, but also in the case of the optic nerves. When, however, it is considered what a large number of injections of arsphenamin are being given every day, and what a comparatively small number of such complications occur, the inference that the drug is the cause is hardly warranted. Mackenzie criticizes the statement, made by some observers, that this affection of the eighth nerve has been caused by arsphenamin, and combats it. He believes that such cases are instances in which the treatment has not been sufficiently active. In the discussion of a paper by Klauder on this provocative action of arsphenamin, read before the Philadelphia

Neurological Society recently, Solomon said that Crockett, of the Massachusetts Eye and Ear Infirmary, had made a study of syphilitic nerve deafness before the arsphenamin era and after, and found the incidence about the same, if anything a little less, since the use of this drug. The absence of the Achilles' reflex on one side in two of the cases is noteworthy in this connection. Beeson [1] in a recent paper refers to abolition of the Achilles' reflex as a danger signal in treatment with arsphenamin. It may indicate a peripheral neuritis due to arsenic.

Under the microscope the appearance of the tissue is distinctly that of syphilis, as shown by the lymphocyte infiltration; but there is also the appearance of alteration of the nerve-fibers which is at least suggestive of the action of a poison.

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1. Beeson, B. B.: Polyneuritis Plus Dermatitis Exfoliativa Following Neo-Arsphenamin, Arch. Dermat. & Syph. **2**:337 (Sept.) 1920.

LATE RESULTS IN EPIDEMIC ENCEPHALITIS*

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There were about 145 cases of epidemic encephalitis in the neurological and medical services at the Mount Sinai Hospital during the epidemics of this disease in 1919 and 1920. A great many of these patients left the hospital soon after the acute infection had subsided, showing some residual symptoms and signs of the disease. After a number of months had elapsed, we felt that it would be of interest to reexamine these patients in order to determine what, if any, changes were still present. With this object in view, this follow-up study was undertaken. Altogether eighty-nine patients were reexamined, and these examinations comprise the basis for this report.

In order to be reasonably certain that none of the symptoms or residual signs found at this reexamination were still part of the acute stage of the disease, at least six months were allowed to elapse before a patient was reexamined. The earliest cases were seen late in 1918, so that some of the patients were examined approximately two years after their acute illness. A complete neurologic examination was made in each case. No attempt will be made in this communication to correlate the symptoms and physical findings in the reexamination with those noted during the acute stage of the illness.

The findings in the cases may be grouped under these headings: (1) psychic disturbances, (2) tremor and irregular involuntary movements, (3) disturbances in attitude and gait, (4) disturbances in tonus and reflexes, (5) residual symptoms in cranial nerves, (6) pupillary disturbances, (7) epilepsy, (8) other residual symptoms and signs, (9) progressive cases, and (10) mortality in these 145 cases.

PSYCHIC DISTURBANCES

Fifty-one of the eighty-nine patients studied showed derangement in their psychic functions in one form or another; twenty-six of these complained of various degrees of irritability; six patients had become so irritable since their illness that they quarreled with everybody who came in contact with them. A number stated that they had no desire

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* From the Neurological Service of the Mount Sinai Hospital, New York City.

* Read at the first annual meeting of the Association for Research in Nervous and Mental Diseases, Dec. 29, 1920.

to associate with any one, and preferred to be alone. They complained of restlessness, were easily disturbed, and would be startled and frightened at the slightest noise. They were unable to concentrate on any subject, and lacked interest in current events. Their mental reactions were retarded, and they were slow in following topics which were being discussed. Slight efforts produced undue physical and mental fatigue. These symptoms were particularly striking in those patients who showed symptoms pointing to involvement of the pituitary or basal ganglions.

Emotional instability was present in many of these patients, and in the majority it was evidenced in a marked depression. They were easily moved to tears, and worried continually—some because they could not sleep, and others because their various symptoms persisted so long after their acute illness was over. A number were worried because they still suffered from drowsiness. A few felt happy and unconcerned; they displayed a feeling of well being that was not in accord with what their physical examination disclosed. Two of these patients were euphoric, and presented a psychomotor activity which was almost equal to that seen in hypomanic states.

There were a number of well-defined cases of fear and compulsion neuroses. One patient, a girl aged 23, was in constant fear that she would commit suicide. She would always have to repress an impulse to jump out of a window, or throw herself before a train. Another patient, a man of 39, was in constant fear of being arrested, even though he felt positive he had done no wrong. A third man was afraid to stay home alone; he did not know what he feared, but felt safe if any one, even a child, would stay in the house with him. He realized how absurd his fear was, but maintained that he could not help or understand it. Another patient, a woman aged 33, could not fall asleep because she could not keep her thoughts away from people she knew who had died. Still another patient could not sleep because many details of the work he had done that day would repeatedly be reviewed.

Disturbance in memory, especially for recent events, was a common complaint. One patient, a student in accountancy, found that since his illness, in addition to defects in memory for recent events, he was having increasing difficulty in handling figures.

Changes in disposition were marked in three children. One, a boy of 10, had a mild attack of encephalitis in March, 1920. Previous to his illness his mother stated that she had great difficulty in managing this child because he was wilful, quarrelsome, and always up to some mischief; since his illness, he has become docile, obedient, and amiable, and never quarrels with his brothers and sisters as he had always done before his illness. Physical examination showed twitching in the

muscles supplied by the upper and lower branches of the right facial nerve and some residue in the other cranial nerves. He had gained twenty pounds; his mental reactions were slow and he resembled a mild type of Fröhlich's syndrome. Another boy of 13 was said, previous to his illness in April, 1920, to be an obedient, well mannered and well behaved child; he never had any difficulty in getting along with his companions. His attack of encephilitis was mild; however, his mother states that since his illness he has become extremely irritable and quarrelsome; he is wilful and difficult to manage. He has great difficulty in his studies and has fallen behind in his school work. He has acquired a spitting tic; every few seconds he has an irresistible desire to spit, and does so accompanying the act with an explosive grunt. He states that he cannot control this desire for more than a few minutes at a time. The third child began to steal little things, and to lie about everything he did; previous to his illness he had been a well behaved child. He has become mentally slow and has difficulty with his school work.

Insomnia was a complaint in forty-nine of the eighty-nine cases; this percentage is so striking that this symptom must be considered an undoubted sequel of encephalitis. Almost all of these patients stated that it required from one to two hours before they could fall asleep, and that until the early morning hours their sleep was easily disturbed; after the early morning hours and during the daytime their sleep was more profound. Some of the reasons given for this difficulty were pains in those patients who had suffered from the radicular type of encephalitis; others, the majority, thought it was due to the fact that they could not relax completely, and that their minds were constantly active. A similar insomnia was noted in many of these patients during the acute stage of their illness. This symptom, therefore, should not be considered a purely psychoneurotic manifestation. Many of the patients complained of excessive drowsiness throughout the day; some of them even fell asleep during their working hours, and were unable to hold their positions on this account. When they went to bed at night, however, sleep was difficult and in some cases even impossible.

Based on the publicity given the disease among the laity, many patients felt that they had acquired a certain importance in having passed through the dangers from it; these patients felt heroic, yet they strongly craved for sympathy and reassurance.

A more extensive analysis of the many variations in the disturbed psychic functions encountered among these patients has not been attempted. I think, however, that sufficient detail has been given to indicate that psychoneuroses and other forms of deranged psychic functions may follow this acute inflammation of the brain. Frank psychoses were not included in this study.

TREMOR AND IRREGULAR INVOLUNTARY MOVEMENTS

Fifty-two patients showed either tremor or some form of irregular involuntary movements. They were present in ten out of twenty-one cases eighteen months after the acute stage of the illness had passed; in eleven of twenty-one cases twelve months after; in twenty-nine of forty-two cases nine months after, and in two of five cases six months after the acute illness. Among these fifty-two patients, there were twenty-five who showed a fine or coarse tremor of either the lips, tongue, facial muscles, head, upper or lower extremities. These tremors closely resembled those seen in toxic states. The finer were like those of hyperthyroidism, and the coarser simulated those seen in chronic alcoholism. Most of the tremors were, as a rule, not evident when the parts were at rest, but became apparent when the parts were put into action. Ten of the fifty-two patients showed a tremor that was spontaneous, present while the parts were at rest, and as a rule limited to the distal parts of the extremity affected. Emotional stress caused exaggeration of the tremor, and it was said to be absent during sleep. This tremor was like the tremor of paralysis agitans. In three cases it was present in both hands; in three others it was present only in one hand; three patients presented tremor in the arm and leg of one side, and in one it was present in both arms and legs.

Tremors of the intentional type were noted in five patients. In three of these patients the tremor was present in both arms; in one the head and both arms were the seat of the tremor, and in the other all of the extremities were involved. These tremors were evident only when the parts affected were in action. They were characterized by large, coarse, irregular oscillations, with a variable range of movement which tended to increase as the goal was reached. They closely resembled the tremors seen in multiple sclerosis and cerebellar disease. One of the patients, in addition to the tremor, had nystagmus, scanning speech, and walked with an ataxic gait. She was, however, improving progressively.

Five patients had a rapid clonic twitch of the muscles supplied by one or more branches of the facial nerve. This twitching closely resembled the muscular response obtained when the nerve is stimulated with an electric current. It occurred every few seconds, was rapid, and always definitely anatomically limited to the muscles supplied by the particular branch involved.

Three of the patients had choreiform movements of the tongue, face or extremities. Three others had fibrillary tremor of the tongue; two of these were associated with a unilateral atrophy of the tongue. One patient had myoclonic movements of the perineal muscles one year after the acute illness.

DISTURBANCE IN ATTITUDE AND GAIT

Fourteen of the eighty-nine patients had disturbance in their attitude or gait, four patients—children—had the typical attitude and gait of paralysis agitans. The youngest was 7½ and the oldest 16 years. Five other patients showed similar attitudes and in addition walked with a hemiplegic gait. Loss of associated movements was present in five of these nine patients. Propulsion was present in four, and in one of these retropulsion was also obtained. Three patients walked with a spastic paraplegic gait and two walked with a right hemiplegic gait.

DISTURBANCE IN TONUS AND REFLEXES

Muscle tone was disturbed in seventeen cases. In four of these there was slight increase; in five there was moderate increase, but not sufficient to produce the "cog-wheeel" phenomenon; in five others there was a marked increase, and the "cog-wheel" phenomenon was elicited. In two patients tonus was diminished in the muscles of the lower extremities, and one patient showed well-marked dystonia in the muscles of both thighs.

The deep reflexes were altered in twenty-seven cases; in eleven of these patients, the deep reflexes were greatly increased, but equally so on both sides. In fifteen the deep reflexes were unequal, being more active on one side than on the other. In one case, the right ankle reflex was absent, and the left was greatly diminished, while both knee reflexes were exaggerated.

The Babinski phenomenon was definitely present in six cases, and doubtful in one. In two of the six patients it was present on both sides, while in the four others it was present only on one side. Three of these patients presented clinically paralysis agitans features. In one of the patients there was associated with a bilateral Babinski sign, hypotonus at the ankles and loss of the right ankle jerk.

CRANIAL NERVE AND PUPILLARY DISTURBANCES

Some disturbance in the cranial nerves was present in fifty-seven of the eighty-nine patients. In thirty-six of these slight facial inequality was noted on one side; two others showed bilateral facial weakness. The slight disturbance in facial enervation in the majority of the cases seemed to be supranuclear in origin. Nine of the patients had both facial and external rectus involvement; two implication of the external rectus alone. Two had atrophy of the tongue and one had fibrillary tremor without atrophy. One patient had deviation of the tongue to the right, and one had paralysis of the soft palate on the right side. Five had some disturbance in the optic nerves during the acute stage of their illness. In this reexamination in only one patient definite changes in

the optic nerves were found; this girl had a temporal pallor on the right side and almost complete atrophy of the left nerve head. No optic neuritis was noted during the acute stage of this patient's illness. She also complained of diminution of hearing on the left side, but there were no objective signs of disturbed function present.

Incomplete ptosis of one eyelid was present in sixteen cases; on both sides in seven others. The pupils were unequal in twenty-six cases. Irregularity of the outline of the pupils was notetd in five. Disturbance in the reflex to light or convergence was present in twenty-six cases; in ten of these there was a sluggish and incomplete reaction to light in both pupils; in seven others only one pupil was affected. In four patients there was a sluggish reaction to light on one side and complete loss of reaction on the other, with sluggish reactions to convergence in one or both pupils. Argyll Robertson pupils were present in five cases; in three of these it was present on one side only, and in two on both sides. One patient who had atrophy of the optic nerves, had what appeared to be on rough testing, a left homonymous hemianopsia, and the pupils were sluggish.

EPILEPSY

Three patients suffered from grand mal or petit mal attacks since their acute illness. The patient with the grand mal attacks had, since his acute illness in January, 1920, four seizures in which there was complete loss of consciousness for a period of from five to ten minutes. He also had a great many seizures in which he did not lose consciousness. The other two patients, both men, one 27 and the other 48 years of age, have been suffering since their acute illness from attacks of spasmodic contraction of the right side of the face. These attacks come on at frequent intervals, last from thirty to sixty seconds, and in the older of the patients are accompanied by vertigo, confusion and difficulty in speech which lasts from one to two minutes. If the men are standing when the seizure occurs they must grasp some near-by object to keep from falling. Any number of attacks occur each day.

OTHER RESIDUAL SIGNS AND SYMPTOMS

Headache and generalized pains in the various parts of the body was a most common complaint. The headaches presented no special characteristics; they were located over any part of the cranium, and were as a rule diffuse. Burning on the top of the head and behind the eyes was frequently an annoying sequel. Pains in the body and extremities was a frequent complaint of those patients who had suffered from the radicular form of encephalitis. Some of these patients stated that their pains were as severe as during the acute stage of their illness;

most of them, however, felt that their pains were gradually getting less severe.

Exophthalmos was noted in four cases. It was present on both sides, and was unaccompanied by any other signs of hyperthyroidism, with the exception of tremor of the hands. Tremor of the hands, however, was present in so many of the patients that it could not definitely be attributed to hyperthyroid activity in these patients.

Increase in weight sufficient to be noted by the patient or his people was found in fifteen cases. In thirteen of these the amount gained above the best previous weight was 15, 15, 17, 20, 20, 25, 25, 35, 38, 45, 48, 50 and 95 pounds. In two the exact amount gained was not definitely known. Many of these patients showed some other manifestations of disturbed pituitary function.

PROGRESSIVE CASES

Seven of the patients gave definite evidence of progression in their disease at the time they were examined. Five of these were of the paralysis agitans type. They showed the typical attitude, gait, tremor, rigidity, and reslessness seen in this disease. In some of these, disturbance in associated movements was also present. One of the other two patients, a man 30 years old, has gained ninety-five pounds in weight since his illness sixteen months ago; he is mentally sluggish and always drowsy; six months ago fibrillary tremor and atrophy of the left half of his tongue were noted. He is progressively getting worse. The other patient was taken ill in November, 1919. At the time her physical examination revealed bilateral facial weakness, tremor of the tongue, intention tremor and rigidity with cog-wheel phenomenon in both arms; her attitude and gait were that of a patient with paralysis agitans. In December, 1920, reexamination showed that she walked with a peculiar gait, not unlike that seen in progressive torsion spasm; there were choreiform and choreo-athetoid movements of the face, neck, shoulders and lower extremities. There was a constant uncontrollable grinding of her teeth. While seated, her pelvis and lower extremities were constantly being twisted by involuntary spasms of the muscles. Her pupils were irregular and almost completely immobile in their reactions to light and convergence. There was slight flattening of the facial folds on the right side. Dystonia was present in the muscles of the lower extremities. The deep and superficial reflexes were present and equally active on both sides. The Babinski sign was not elicited.

The majority of the other patients admitted that they were gradually improving. Even though they still had many complaints, they were much better and the symptoms were far less severe than they had been at the time the patients were in the hospital.

MORTALITY

Among the 145 patients admitted to the hospital twenty-nine died, giving a mortality rate of 20 per cent. Notice was received of the death of one other patient after she left the hospital.

SUMMARY AND CONCLUSIONS

Sufficient time has not yet elapsed since the acute illness, nor is the number of cases in this study large enough, to warrant drawing absolute conclusions as to what the ultimate prognosis will be in these patients. There are, however, a number of striking facts that might be emphasized.

1. Psychic functions in some form or another were disturbed in 55 per cent. of these patients.

2. Insomnia was present in 55 per cent. of the cases.

3. Tremor and irregular involuntary movements were found in 58 per cent. of the cases.

4. The deep reflexes were altered in 30 per cent. of the cases, and tonus in the muscles was disturbed in 18 per cent.

5. The cranial nerves showed residual signs in 64 per cent. of the cases.

6. Pupillary disturbances were found in 30 per cent of the cases. Five patients had Argyll Robertson pupils.

7. About 8 per cent. of the patients gave signs of progression at the time they were examined.

8. The mortality among the 145 patients admitted to the Mount Sinai Hospital was 20 per cent.

From these findings one might venture this tentative prognosis: Probably less than 20 per cent. of the patients who become ill with epidemic encephalitis die during the acute stage of the illness, as usually only the most severe cases reach the hospital. Of those who survive the acute stage, about 10 per cent. may develop a progressive disease of the central nervous system. The remainder will make a good functional recovery in from six to twenty-four months, with the probability of progressive approach to the normal after that period.

News and Comment

CENTENARY OF BLOOMINGDALE HOSPITAL

The hundredth anniversary of the opening of the Bloomingdale Hospital will be celebrated on May 26. The exercises will include addresses by Dr. Pierre Janet of Paris, Dr. Richard G. Rows of London, Dr. Lewellys F. Barker and Dr. Adolph Meyer, Baltimore. The hospital is the department of nervous and mental diseases of the New York Hospital which, since it was opened in 1792, has made provision for the treatment of persons suffering from mental disorders. As early as 1808 a separate building was provided for these patients at the general hospital at Duane Street and Broadway, New York. In 1821, the department was removed from the city to a point on the Bloomingdale Road which is now the location of the library of Columbia University, and was given its present distinctive name. Since 1894, it has been located at White Plains.

Abstracts from Current Literature

LA MICROGLIA Y SU TRANSFORMACION EN CELULAS EN BASTONCITO Y CUERPOS GRANULO-ADIPOSOS (MICROGLIA AND ITS TRANSFORMATION INTO ROD CELLS AND GRANULO-ADIPOSE BODIES). P. DEL RIO-HORTEGA, Arch. de Neurobiol. **1**:171 (June) 1920.

The author summarizes the article thus:

1. Relation between microglia and the third element (of Cajal).

2. Characters of normal microglia: (*a*) technic; (*b*) morphology of the microglia; (*c*) morphologic variations in normal states; (*d*) distribution and relations of the microglia.

3. Abnormal characters of microglia: (*a*) first morphologic changes; (*b*) the problem of rod cells; (*c*) formation of rod cells; (*d*) character of the rod cells; (*e*) problem of granulofat cells; (*f*) formation of granulofat cells.

4. The probable nature of the microglia.

The author gives a résumé of the views of Alzheimer, Held, Fieandt, Jacob, Lugaro, and others, who have denied the existence of a so-called third element of the neuroglia, and of Bevan, Lewis, Nissl, Robertson, Bonome, Schaper, Rosenthal, and others, who have considered the third element as undifferentiated glia without dendrites, and have thought that they had observed transitional forms between the adendritic bodies (third element) and dendritic glia cells. He then gives Cajal's conclusions as to a veritable third element, at length, resulting in the classification of the third element into two groups, subdivided into several subgroups; the two groups being (*a*) cells of the gray matter, perineural satellites not neuroglial, and disseminated apolar cells; (*b*) apolar cells of the white substance, either isolated or arranged in rows.

The author then passes to his own methods of research which have been carried out on the brains of monkeys, rats, dogs, cats, rabbits, oxen, goats and sheep; on linnets, pigeons and hens; lizards, newts, snakes, chameleons and turtles; toads and frogs; and many fish and above all on human brains of all ages and of people dying of ordinary diseases.

In general, microglia is described as consisting of very small corpuscles provided with large, ramified expansions. It is diffused throughout the nervous tissue, but differs in character in varying localities. It differs from neuroglia in not possessing the characters of protoplasmatic glia, reticulum, gliosomes, nor those of fibrous glia, differentiated fibrils. Centrosomes and Golgi's net are also absent in microglia.

Morphologically the microglial cells are:

(*a*) Monopolar with dendrites ramified not far from the body.

(*b*) Bipolar with two thick dendrites with many right-angled collaterals, the principal dendrites often ending far from the cell in a plume of very delicate branches, often double nucleated.

(*c*) Multipolar, most numerous of all and offering great variety of form.

The microglia is present in all parts of the brain in greater or less quantity, but much more in the gray matter than in the white.

In the gray matter the greater number of neuronal satellites are probably microglial. The vascular satellites are also greater in number in the gray than

in the white substance. These vascular satellites do not enter into intimate relations with the vessels as in the glial feet of neuroglia. Microglial cells are also seen as satellites of neuroglial cells.

These morphologic characters are identical in man and the vertebrates studied, differing in this manner from neuroglia which shows marked differences in the different groups of vertebrates.

The first pathologic change which takes place in microglia is an increase in volume, especially of the dendrites; sometimes hypertrophy of all the microglia occurs. This change is seen in man, especially in acute and subacute meningitis, meningo-encephalitis and uremia, but seen to a certain extent in many other pathologic states. Shortening of the dendrites occurs coordinately with their thickening; argentophile granules are seen, but none colored by scarlet.

The author considers the various theories of the origin of rod cells, describes them as characterized by a long, rectilinear, curved, round, ovoid or fusiform nucleus, pale protoplasms, orientation almost always parallel to blood vessels and perpendicular to the surface of the cortex. He notes atypical forms. He considers the rod cells as phagocytes and goes into detail as to their formation from emigrating microglial cells, with the same minutia as he studies the history of the granular cell and finally describes the formation of granular cells from the microglia.

His conclusions are:

1. Besides the neuroglia a third type of cells exist whose character and origin have given rise to much argument.

2. Two forms of neuroglia have been isolated, one protoplasmatic with short dendrites especially proper to the gray matter, the other fibrous with long radiations proper to the white substance.

3. The third element has been considered as an interstitial element characterized by the absence of prolongations which would take the elective glial stains.

4. In such cells, apparently apolar, two varieties have been recognized, one with a large clear nucleus, the other with the nucleus small and dark colored.

5. The author's observations confirm the actuality of both types of cells mentioned in the foregoing, but of a character entirely different to what has been assumed hitherto. The two types are (*a*) interfascicular glia and (*b*) microglia.

6. The interfascicular glia, which is situated by preference between the nerve fiber bundles of the white substance, although also accompanying nerve cells and blood vessels, is characterized by a large vesicular nucleus, a round or polyhedral body, by its epithelial appearance and by scanty, long, filiform prolongations, little branched.

7. The microglia is seen throughout the nervous tissue, more abundant in the gray than in the white matter, and is characterized by its small dark nucleus surrounded by little protoplasm, and by its long, tortuous expansions which are much ramified and provided with lateral spines. Neither gliosomes nor gliafibrils are seen in its obscurely reticulated protoplasm, but pigment and lipoid granules are frequently seen in its substance.

8. According to its situation, the microglia is classified as a neuronal, vascular and neuroglial satellite.

9. No morphologic or structural differences exist in the microglia of different vertebrates.

10. This constancy of character is due principally to the fact that the microglia is subjected to the general disposition of the nervous system between the interstices of which the prolongations of the microglia are inserted. Therefore, the forms seen are due to adaptation to the surrounding mediums.

11. The instability of form of the microglia is seen more in embryonal development and in the pathologic conditions than in adult and normal brains.

In their evolution the microglial corpuscles pass from a rounded to a stellate form, and in regression they tend to recover the original form.

12. Judging from its action in pathologic conditions, the microglia is especially fitted for phagocytic action on the degenerative products of disintegrating nerve cells.

13. When the microglia is put into movement and enters on its phagocytic action in consequence of some disturbance of the nervous system, its form is modified little by little, and the microglia is transformed into rod cells and granulb-adipose cells.

14. The long bipolar cells (rod cells) appear only when the microglia extends parallel with fiber bundles, or nerve cell dendrites. Ordinary methods of staining show these cells only in pathologic processes of slow development. In acute processes the bipolar types regress to the multipolar type or are transformed into the more or less rounded granular type.

15. In order to become rounded (granular cells) the microglia cells must be freed from the pressure of the surrounding structures, as by liquefaction or rarefication, as well as filled with degenerative products.

16. The forms which the microglia takes on in pathologic processes are similar in form, structure and coloring qualities to those of the emigrating connective tissue corpuscles in abscesses, tumors, etc.

17. There are many reasons for considering the microglia mesodermic in origin and, if confirmed, the name mesoglia would be justified.

18. The microglia is, therefore, a third element of the nervous centers. The interfascicular glia appears to be a third type of neuroglia of epithelial origin.

The author notes that during the preparation of the present article he has made important observations to be published fully, later, which tend to prove the mesodermic origin of the microglia.

As usual in the work of the Spanish school, the illustrations are very profuse, excellent and conclusive.

GURD, Ann Arbor, Mich.

DIE PLEUS CHOROIDEI BEI ORGANISCHEN HIRNKRANKHEITEN UND BEI DER SCHIZOPHRENIE (THE CHOROID PLEXUS IN ORGANIC DISEASES OF THE BRAIN AND SCHIZOPHRENIA).

SADAMICHI KITABAYASHI, Schweiz. Arch. f. Neurol. u. Psychiat. **7**:1, 1920.

Comparatively little work has been done on the study of the choroid plexus in the organic diseases of the brain, and its physiologic significance is comparatively unknown. The present contribution is a continuation of the study of the choroid plexus, several reports of which have been abstracted in these columns. Previous investigations of the brain of schizophrenic patients have shown that the choroid plexus is subject to characteristic alterations in this disease. Degeneration productions of various kinds, which cannot be more closely identified, are numerous. Some of these penetrate the ependymal wall into the subependymal structures and even into deeper tissues of the brain, probably as a result of abnormally low resistance of the ependyma.

The present report was based on the study of three groups of cases. The first group is made up of five patients whose ages ranged from 11 months to 55 years, whose brains were assumed to be practically normal. The second group of eight patients represents those who had chronic diffuse or localized lesions of the central nervous system without schizophrenic manifestations. The third group is composed of patients who had schizophrenia.

Kitabayashi studied not only the choroid plexuses themselves, but also the adjacent ependymal walls. The material was fixed in formaldehyd, dehydrated with alcohol, and embedded in celloidin; sections 15 microns in thickness were stained with hematoxylin, van Giesen, toluidin blue, and carmin. Some of the material was embedded in paraffin.

The normal plexus was found to differ somewhat in the microscopic appearance, depending on the age. In children and young persons, the vessels of the plexus are very delicate throughout; the connective tissue is small in amount, very delicate in structure, and free from large spaces. The glandular cells have a grapelike arrangement near the base of the villi and like a string of pearls along the free portion, there being but one row of cells here. The protoplasm is finely granular throughout, free from definite tigroid elements, fills the cells completely, and contains a well defined, deeply staining nucleus which contains a nucleolus. Vacuoles are not seen. There are no colloid masses, no hyaline, and no calcium deposits anywhere. The appearance of the plexuses in the different ventricles is essentially the same.

The above description, in the main, applies to all normal cases; in old persons, however, there are slight modifications. In a patient 30 years of age, the perivascular spaces were still small and delicate in structure as compared with those found in older persons. Only occasionally was a nucleus seen that was somewhat shrunken. Here and there an area was found in which the connective tissue was somewhat thickened and showed slight hyaline degeneration. At the age of 35, in addition a few calcium granules were noted. These were numerous in the perivascular spaces of a man 55 years of age. The plasma and nuclei of the glandular cells were slightly atrophic.

Since the patients of the second group showed changes that were not uniform, it is necessary to review them individually. The first was a premature baby of about 7½ months, who had hydrocephalus and died of convulsions. Necropsy revealed, in addition to the hydrocephalus, microgyria and almost complete absence of the corpus callosum. The villi of the choroid plexuses were small (microplexia). The cells themselves had the appearance of atrophy, possibly the result of pressure. In the connective tissue spaces, a few albuminoid granules were noted, which is unusual for normal children of the same age. The ependyma showed an alternating atrophy and hyperplasia; the subependymal tissue was hyperplastic, probably the result of pressure or penetration of noxious substances contained in the spinal fluid.

The second patient was a taboparetic, 42 years of age, without hallucinations and delirium, who died suddenly in a status epilepticus. The plexus showed little that was characteristic; there was slight swelling of the cells and a few small vacuoles were noted. There was a slight exudate which, however, was entirely free from the granules that are so characteristic of schizophrenia.

The third patient of this series was a man of 61 years, who had had right-sided jacksonian epilepsy for a number of years, and headaches. Examination revealed choked disks, a right-sided hemiplegia, dysarthria and motor aphasia. There were no marked psychic alterations. A decompression operation fol-

lowed. Death occurred suddenly at the end of seven years. Necropsy revealed an angiosarcoma in the left central convolution. The plexuses showed a dense increase in the connective tissue of the perivascular spaces and some hyaline degeneration of the plexus cells.

The fourth case was that of a teacher, 72 years of age, who, following fracture of the tibia, developed a marked psychosis with dementia, confabulation, delirium, hallucinations, complete disorientation—on the whole, a manic picture of several months' duration. The plexuses revealed changes ordinarily noted in senile patients, namely, an increase in connective tissue and deposits of calcarious concretions; in addition, there were findings not characteristic of senility, such as marked degenerative changes of the plexus cells, which were swollen and showed ameboid processes, vacuolization and desquamation. The exudate in the intervillous spaces contained albuminoid granules that could be traced into the subependymal tissue where they doubtless acted as foreign substances. The writer believed that there was probably a close relationship between the pathologic findings and the clinical manifestations.

The fifth case was that of a weaver, 65 years of age, in whose case a diagnosis of an organic psychosis with manic-depressive features was made. The patient showed increased psychomotor activity with impulsive acts, visual and auditory hallucinations, and sitophobia. The clinical picture approached that of schizophrenia. The villi of the plexuses were markedly degenerated; there was sclerosis of the cells, reduction in size of the nuclei, desquamation of entire rows of cells, massive concretions within the villi, and numerous albuminoid masses that could be followed into the subependymal tissue. There was also a heterotoxia of the vermis. The writer thought that here also the anatomic findings might explain the psychic processes.

The sixth patient was a porter, 47 years of age, who was a chronic alcoholic, and who had suffered a fracture of the spine. He had hallucinations, delusions of persecution and marked disorientation. He died of pneumonia. The vessels of the villi were degenerated; there were perivascular extravasation of the blood and atrophy, sclerosis, swelling, desquamation and vascularization of the choroidal cells. The writer believed that the parenchymatous and hyaline changes of the capillaries could be attributed to alcoholism; capillary thrombosis and blood extravasation followed the vascular degeneration. The exudation between the villi might have resulted from fever.

The seventh patient was a 70 year old man, who suffered from arteriosclerotic dementia with epilepsy, illusions and delusions of persecution. At necropsy, senile changes were noted in other parts of the body and evidenced in the choroid plexuses by sclerosis, tortuosity and thrombosis of the arteries, and marked enlargement of the perivascular spaces, which contained dense calcarious concretions. These changes may be attributed to the arteriosclerosis. There was also desquamation of the cells, the latter being in part atrophic and sclerotic, and in part swollen and vacuolated. The connective tissue contained numerous albuminoid bodies, some of which penetrated into the subependymal structures.

The last patient was a man 53 years of age, a congenital deafmute and an idiot. The plexus cells were moderately atrophic—some were strikingly small, others sclerotic or swollen and still others desquamated. There was definite connective tissue increase about the blood vessels; hyaline degeneration and calcified granules were noted in the villi. These findings were probably the result of age. On the other hand, in the spaces between the villi albuminoid

bodies were noted which also penetrated into the subependymal tissue. The vermis was heterotaxic; the ventral acoustic ganglion and the tuberculum acusticum were greatly degenerated, which probably explained the deafness.

The article will be continued.

WOLTMAN, Rochester, Minn.

LE HOQUET EPIDEMIQUE, FORME SINGULTEUSE DE L'ENCEPHALITE EPIDEMIQUE (EPIDEMIC HICCOUGH, A SINGULTOUS FORM OF EPIDEMIC ENCEPHALITIS). J. LHERMITTE, Presse méd. **28**:916 (Dec. 18) 1920.

The epidemics of hiccough noted by Boerhave at the hospital of Haarlem, that of the convent of Monterrey (Spain), also those reported by the medieval historians and attributed to demon-possession, formerly found a fully satisfactory explanation in the theory of hysteria. Recent epidemics of hiccough have proved that even if certain cases do arise from suggestion or psychic contagion, there are others for which another explanation must necessarily be found.

Von Economo reported the first appearance of epidemic hiccough at Vienna in the winter of 1919-1920, as follows: "Several weeks before the appearance of this epidemic of encephalitis in Italy (January, 1920), there was a small epidemic of singultus in and near Vienna. Numerous individuals were suddenly seized with painful attacks of hiccough, without any preliminary symptoms of illness; these attacks lasted hours and even whole days, were not affected by any treatment, and ceased spontaneously at the end of a few days. I saw a similar case that lasted an entire month, the patient having only a few hours of rest each day. This disease had no serious sequelae."

One month after this epidemic of hiccough was over the first cases of myoclonic encephalitis appeared, imitating the variety that had just undergone a violent exacerbation in Italy. The illness commenced with general malaise, accompanied by vague rheumatic pains and often by severe neuralgias. Then an occupation delirium appeared, with visual hallucinations, and finally clonic convulsions of the abdominal muscles, and hiccough. At this point a state of choreiform agitation supervened, followed by myoclonic contractions or fibrillary twitchings, not interrupted by sleep.

In January and February of 1920, Dufour and R. Bénard described a number of cases of hiccough in and near Paris. Typical cases cleared up about the fourth day; one terminated in myoclonic convulsions and death. Staehelin's studies at Basle revealed the successive development, first, of an epidemic of transitory ocular paralyses, then of an epidemic of hiccough, finally of an outbreak of numerous cases of epidemic encephalitis. In November, 1920, epidemic hiccough reappeared at Paris.

In a simple hiccough the abdominal wall is passively lifted, but its muscles do not exhibit spasmodic contraction. In epidemic hiccough the clonic spasms may not be so closely limited to the constrictors of the glottis and of the diaphragm, but may extend to the abdominal muscles and even reach segments of musculature whose rôle is entirely independent of the respiratory function. Clonic contractions added to the spasmodic shocks of hiccough occur most often in the muscles of the back, the nucha, or the limbs. Different attitudes result, as backward bending of the trunk, bowing of the head, flexion of the extremities, which vary with each patient, and in the same person are subject to modifications according to the changing conditions in the nervous system. In rarer cases, the hiccough is associated with spasmodic contractions limited to the abdominal muscles of one side. Achard recently

demonstrated radioscopically a strictly unilateral contraction of the diaphragm in an epidemic hiccough case.

Pain is uncommon. Phonation and deglutition are markedly disturbed. Patients adopt various psychic attitudes: some are greatly distressed and give up all social activity; others "rebel, and while excusing themselves to their associates for this inopportune ailment which they consider ridiculous, make no change in their daily habits." The rhythm of hiccough ranges from six to fifteen contractions per minute, which go on without intermission for hours, sometimes for whole days. The total duration is three or four days, ordinarily, but may be much longer. The onset may occur without any prodromal symptoms, but a dilligent examination or searching anamnesis will often discover general malaise, chilliness and headache, accompanied by slight elevation of temperature to 99 or 100 F. Mild emotional disturbances are often detectable. Visual functions, deep and superficial reflexes, and general muscular tone show no abnormalities.

Epidemic hiccough constitutes a syndrome whose elements, outside of the phrenoglottic myoclonus, are necessarily elusive, fleeting, and variable. It is differentiated from hysterical and even more from simulated hiccough by the fact that certain of its characteristics cannot be reproduced by a normal subject. The frequency of hiccough in abdominal and peritoneal disease has led to a number of surgical operations on patients with epidemic hiccough. Typical lesions of acute encephalomyelitis have been found at necropsy.

Quoting a number of other European and American observers on the frequency of hiccoughs in epidemics of unquestioned "encephalitis lethargica," Lhermitte believes the epidemiologic and clinical evidence is conclusive that epidemic hiccough is only a masked form of epidemic encephalitis. The pathogenesis remains unknown and the therapeutics unreliable. Treatments for hiccough fall into two groups—drugs that counteract bulbospinal and vagophrenic hyperexcitability (belladonna, atropin, cocain, morphin, bromid, camphor, oxygen), and physical means of exercising an inhibitory action on nerve centers in a state of reflex excitement (a dozen of these are mentioned). Their diversity denotes their inadequacy.

HUDDLESON, New York.

BEITRAGE ZUR KLINIC UND PATHOGENESE DER LUMBAGO (CONTRIBUTIONS TO THE CLINICAL STUDY AND PATHOGENESIS OF LUMBAGO). RUDOLF BRUN, Schweiz. Arch. f. Neurol. u. Psychiat. **7**:63, 1920.

Although lumbago is a subject of great practical interest, the serious study of it has been neglected until a comparatively recent date. Even at the present time this banal disturbance is little understood. The clinical findings have been almost uniformly negative.

In all probability, "lumbago" has numerous and diverse pathologic bases. Its tentative division under the three heads of myogenic, osteo-arthrogenic and neurogenic, probably represents impressions and theories rather than well-founded clinical diagnoses.

The myogenic form has been further subdivided. The assumption is that the majority of these cases are rheumatic in nature; this would also apply to most cases in which the symptoms followed an injury, the idea being that these muscles had been rendered more susceptible to traumatism by a low grade myositis, the symptom of pain becoming manifest only on the occasion of an injury. The history of a previous rheumatic condition in some other location

strengthens the diagnosis. Many writers share the opinion that most cases of lumbago represent recurrences of a subacute or chronic lumbar myositis. According to Gelpke, secondary infection of the traumatized muscles explains the usual chronic course of the disease.

The second division includes the actual muscle trauma, such as rupture, tearing of the ligaments and fascial hernias.

The osteo-arthrogenic type is probably rare.

The neurogenic type is supposed to originate from compression of the sensory nerves. The frequent association with sciatica has been explained as a spreading secondary perineuritis.

Brun reports twelve cases of so-called "lumbalgia," in which in each instance objective findings could be demonstrated. Of fifteen cases seen in the Zurich neurologic clinic between 1915 and 1919, only one in five gave negative findings. According to Pometta, one of the best students of the subject, traumatic lumbago should heal within from five to eight days. In these patients, however, the progress was eminently chronic, extending over months and even years. He could almost uniformly demonstrate some abnormality in the region of the lumbar musculature, particularly the erector trunci and the sacrospinalis muscles. In eleven of the twelve cases, inspection and palpation alone gave positive findings. In two cases a transverse groove was found, which was interpreted as a rupture of the muscle; this could be demonstrated more clearly by voluntary movement or by faradic stimulation when it was noted that the upper portion of the ruptured muscle retracted strongly upward while the lower portion did not respond. In the ten remaining cases the lower insertion of the sacrospinalis muscles appeared definitely depressed, atrophic, inelastic, tender and in every instance gave abnormal electrical responses. Irritability to the faradic current was diminished or did not result in mass contraction. Of seven cases tested with the galvanic current, only two gave normal responses, while the remaining five showed a partial reaction of degeneration. Abnormality of the vertebral column was noted in some. Tuberculosis of the spine was disclosed by the roentgen ray in a patient whose case had for a long time been diagnosed as lumbago; the roentgen-ray findings in the others were negative. Tenderness of the spine was not uncommon. Scoliosis, when present, was usually directed toward the healthy side. In several cases, there was a spasmodic reflex fixation of the lumbar vertebrae on bending. Hyperesthesia and hypesthesia were noted in the area supplied by the posterior roots of the lumbar and sacral nerves, the ileo-inguinal and the sciatic. Reflex irritability of the sympathetic supply was evidenced by cutaneous flushing on slight irritation. In three cases there was an associated neuralgia of the ileo-inguinal nerve, in one of which an associated spermatorrhea improved coincident with the recovery from the lumbago. Tenderness on pressure over points at which these nerves pierce the fascia was common. The lower abdominal reflexes were often reduced while the cremasteric reflexes were increased. In all but one patient there was referred pain along the ipsolateral sciatic nerve, although the characteristic signs were usually absent.

Brun reviews somewhat extensively the sympathetic innervation of muscles and the difference in metabolism of the two components of muscle fibers as described by Boeke, Pekelharing, de Boer and others. Their work is applied to the question of lumbago in so far as he assumes that practically all of these patients are neurotic; their sympathetic innervation is therefore altered, and in such a manner that the musculature loses its ability to with-

stand sudden stresses, thus becoming more fragile and susceptible to rupture. Ischemia also plays a part in this.

He calls attention to the fact that a nerve may easily be injured in its intervertebral course by a transient subluxation of the spine, which may at once correct itself, while the injury to the nerve persists. Extension to the sciatic and ileo-inguinal nerves usually does not occur until several weeks or months after the initial trauma. This radiation, he states, takes place through the spinal ganglions and is dependent on the increased irritability of the sympathetic cells as well as on over-irritation of the remaining uninjured fibers.

The frequent chronicity of the disorder and its association with a general neurosis can be demonstrated by psychanalytic methods, to rest on a sexual basis, the lumbago becoming the peace offering onto which compunctions of conscience arising from onanism and impotence can be heaped.

Brun's conclusion is that lumbago is usually due to perineuritis of traumatic origin and seldom rests on a toxicinfectious basis. He emphasizes the importance of a careful study of the individual case.

WOLTMAN, Rochester, Minn.

INTELLIGENCE AND PSYCHOSIS FROM THE PSYCHOLOGICAL LABORATORY OF McLEAN HOSPITAL, WAVERLEY, MASS.

F. L. WELLS and C. M. KELLY, Am. J. Insan. **77**:16, 1920.

One hundred and two pàtients, both men and women, ranging in age from 16 to 75 years, suffering from mental disease, various psychoses being represented, were examined according to the Stanford Intelligence Scale, and the results were tabulated according to diagnostic groups. Analysis showed: 1. Reduction of intelligence to subnormal was not a necessary accompaniment to grave mental disorder. Seventy per cent. of the patients had intelligence quotients above 70 and except for the organic group, over one half presented intelligence quotients above the level to which independent adjustment is for reasons of intelligence no longer possible, and some were considerably above the normal level. 2. Little diagnostic importance could be attached to intelligence quotients, but the organic cases showed the greatest reduction, being the only cases in which intelligence defect showed definite association with the psychosis. 3. The greatest amount of "scattering" was found in the organic group, less in the dementia praecox and least of all in the manic-depressive group, but the difference between the groups was small. A few single tests were the only differential features. For example, absurdities (X-2), designs (X-3), reading and report (X-4) were never passed above age, but failed with similar frequency in the three groups. The superior plan, ball and field (XII-3), failed below age over twice as often in the dementia praecox group as in the manic-depressive or organic group, while repetition of five and six digits backwards (XII-6, XVI-5) resulted in nine failures below age, two successes above in the manic-depressive and organic groups with one failure below age and eight successes above in the dementia praecox group. It was also noted that several of the tests appeared to be differently weighted for the insane, that is, they did not have the same value for the psychotic as for normal persons of a given mental age. Designs (X-3), reading and report (X-4), and ball and field, superior plan (XII-3), were much more difficult for these subjects, showing a marked tendency to failure below mental age, while vocabulary and formal memory tests were the least difficult and the most frequently passed above mental age.

Some criticism of detail was offered, principally defects inherent in the scale, especially true when used for patients of this class—but the scale was not constructed for this group. Among the most noticeable were the childish phraseology of many of the tests, problems which presented situations incongruous with adult experience or of limited application because of local and geographic differences. Several of the tests lent themselves most readily to coaching and were therefore found just that much less valuable. Duplication of digits and sequences in the number series for memory tests were objected to on psychologic grounds. Other tests allowed scope for perseveration. Lastly, the emotional setting of certain of the tests was disturbing to some patients and influenced results, while others allowed for perverted responses, most conspicuous in the dementia praecox group. It was further noted that "intelligence" as measured by the scale was not all-inclusive in that ability to deal with ideas is emphasized while no cognizance is taken of ability to deal with things or make adjustments to other persons, both equally important in "real" action. Intelligence in this limited sense is neither the sole nor the chief factor in practical mental adjustment but is essential in some degree. In so far as the scale measures ideational capacity, it was found to be satisfactory and practical but with the psychotic groups the failures of adjustment for the most part are not in the sphere of ideas or things but in the more instinctive and affective adaptations to other members of society. Similar intelligence quotients, therefore, appeared in extremely differing personalities and normal ones in the presence of grave mental imbalance.

PERKINS, Detroit.

TRAITEMENT DE LA SYPHILIS NERVEUSE PAR LES INJECTIONS NOVARSENICALES A PETITES DOSES REPETEES ET PROLONGEES (TREATMENT OF SYPHILIS OF THE NERVOUS SYSTEM BY PROLONGED COURSES OF FREQUENT SMALL INJECTIONS OF NEO-ARSPHENAMIN). J. A. SICARD, Presse méd. **28**:281 (May 8) 1920.

One of the common neurologic schemes of intensive arsenical medication is a course of from four to six weekly intravenous injections, often in ascending doses, as 0.3, 0.45, 0.6, 0.75 gm. Some workers have tried reducing the dosage and lengthening the interval. This paper presents arguments against these plans, and sets forth the advantages of the following one.

A neo-arsphenamin product ("novarsénobenzol," "sanar," and "galyl" are mentioned) is injected, either intravenously or subcutaneously. Subcutaneous doses do not exceed 0.15 gm., dissolved in 1 c.c. of sterile distilled water. The preferred site of injection is the upper third of the thigh; little pain is experienced and no complications need be feared. Some patients have received without ill effects, as many as sixty daily subcutaneous injections, alternating between the right and left subtrochanteric areas.

The total dose of neo-arsphenamin for general paralysis reaches an average of from 28 to 30 gm. in a year, divided into 9 or 10 gm. for a four month period, 0.15 gm. being given in daily intravenous injections. For progressing tabes, from 20 to 25 gm. are given over a year's time, divided into 6 or 8 gm. for a four month period, two subcutaneous injections of 0.15 gm. and one intravenous of 0.3 gm. being given weekly. For spastic paraplegia, either the tabetic or the paretic outline is followed. The total quantity of drug in any course is reduced for appropriate indications.

This method dispenses altogether with mercury and iodid preparations, and avoids toxic effects peculiar to them. It is claimed that it also avoids the more serious accidents sometimes associated with other methods of neo-arsphenamin administration. Four milder types of reaction are observed: erythema, loss of the Achilles' reflex, late jaundice and slight transitory nitrogen retention.

Two varieties of arsenical erythema are distinguished. One appears early, in 6 or 8 per cent. of cases, after 0.5 to 3 gm. of neo-arsphenamin have been administered; it disappears readily and is generally insignificant. The other appears late and constitutes positive evidence of arsenic saturation. It will progress rapidly through severer stages of skin and constitutional disturbance, unless the drug is discontinued immediately. Treatment is not to be resumed for eight or ten weeks thereafter. This erythema shows an incidence of 2 or 3 per cent.

Disappearance of the Achilles' reflex is discussed at length. This phenomenon attains the remarkable incidence of 60 per cent., but it must be remembered that a course of treatment totals from 7 to 10 gm. of neo-arsphenamin. Incipient objective signs can be detected after 3 or 4 gm. have been administered, and their evolution follows until both ankle jerks disappear completely, during about the sixth or seventh week of the course. As a rule, no subjective weaknesses nor sensory disturbances are experienced by the patients, but paresthesias of the soles and great toes appear occasionally. Several cases have been observed over one and one-half years, and no abolished ankle jerk has reappeared. However, no loss of muscular power supervenes. Syphilitic spastic paraplegias are especially benefited by this fact. A diminishing ankle jerk may go on to complete disappearance one or two weeks after medication ceases. One patient of 200 treated developed a polyneuritis after receiving 4 gm. of neo-arsphenamin.

Jaundice appears in 2 per cent. of cases. It is considered to be of toxic arsenical origin, and calls for discontinuing medication. Uneventful recovery takes place three or four weeks later. Nitrogen retention is of similar import.

Blood and spinal fluid Wassermann reactions, as well as clinical symptoms, are said to be most effectively influenced by this method. No objective signs of intoxication have been discovered in visual, auditory or other sensory fields. The theoretical possibility of producing an arsenic-resisting strain of spirochetes is set aside as already disproved by certain Pasteur Institute work (not cited in detail). Final conclusions are stated moderately: "Comparing equal quantities of neo-arsphenamin injected throughout equal periods of time, one finds that small doses given daily or every other day afford a degree of certainty and safety that cannot be claimed for weekly injections."

HUDDLESON, New York.

ACUTE PSYCHOSES ARISING DURING THE COURSE OF HEART DISEASE. DAVID RIESMAN, Am. J. Med. Sc. **161**:157 (Feb.) 1921.

This is an interesting presentation, from the standpoint of the internist, of the acute psychoses and psychotic symptoms, arising in heart disease. Probably the most common mental syndrome is a state of irritability with a substratum of emotional depression which often occurs in association with cardiac decompensation. A frequent cardiac-psychotic symptom is an hallucination (?) of sight or hearing, which appears in the course of aortic disease. "The hallucinations are to be looked upon as misinterpreted sensations caused either

by anemia or by venous stasis of definite brain centers, those connected with sight or hearing, or by circulatory or trophic disturbances in the corresponding peripheral end organs." If the author's theory is true and we are really dealing with a misinterpreted sensation, it would be better to speak of an illusion and not an hallucination.

Riesman also calls attention to the period of confusion noted just prior to, or just after, sleep in patients with myocarditis and auricular fibrillation; to the mental excitation and marked disorientation in elderly patients with fibroid myocarditis, which is of grave prognostic import; to the abrupt maniacal outbreaks, which occurred in acute pericarditis and in advanced decompensation of myocardial origin, and to the paranoid delusional trend in conjunction with aortic disease.

There are a number of etiologic possibilities: In a person whose heredity is heavily charged, the psychotic manifestations may be entirely independent of the cardiac pathology; kidney disturbance, and more particularly, uremia may be accountable; acidosis may be the precipitating agent or unwise digitalis therapy may "by disturbing cardiac rhythm through its action on the conducting mechanism, still further impair an already inadequate circulation" and thus produce mental symptoms. In the absence of any of these factors "we may assume some direct disturbance of the cerebral circulation, affecting the higher centers." In this connection Dana's[1] theory of "hypofunction of synapses" may condition the abnormal psychotic response to the somatic disease.

From the standpoint of the psychiatrist, our knowledge must be said to be still in the formative stage. We must first be given more light on the chemistry of heart disease and indeed of somatic disease in general. Clinically, it is probably true that the more widely the mental symptoms depart from the classic psychoses, the more readily and accurately may they be attributed to cardiac disease. In other words, we cannot speak now of a "cardiac psychosis" and describe a definite clinical entity though, as Riesman points out, there is certainly a syndrome of mental symptoms which is often associated with cardiac pathology. We have observed the course of a chronic psychosis complicated by added mental symptoms, such as affective depression, which it seemed fair to attribute to heart involvement; on the other hand, patients with serious cardiac conditions often in the decompensation stage may present a typical mania or depression, apparently uninfluenced by the somatic disease.

STRECKER, Philadelphia.

ZUR KENNTNISS DER GANGLIO-ZELLULAEREN HIRNGESCHWUELSTE (A CONTRIBUTION TO FURTHER THE KNOWLEDGE OF GANGLIOCELLULAR BRAIN TUMORS). R. HERMANN JAFFE, Virchows Arch. f. path. Anat., Supplement to **227**:27 (March) 1920.

The author remarks on the difficulty in differentiating proliferated glia cells from ganglion cells, and on the unreliability of the older communications in regard to this subject. Schminke was the first to describe a typical ganglioneuroma of the brain and Pick, Bielschowsky, Achucarro and Robertson have since described cases of ganglioneuromas made up of more or less typical, often enormous, ganglion cells, nerve fibers and glia elements.

1. Dana, C. L.: Somatic Causes of Psychoneuroses, J. A. M. A. **74**:1139 (April 24) 1920.

Large cells have also been found in Strümpell-Westphal's pseudosclerosis and in tuberous sclerosis of the brain. Alzheimer described giant glia cells in pseudosclerosis which degenerate without any tendency to form fibers. Many of the cells were almost filled with large nuclei with irregular outlines and projections. In tuberous sclerosis identification of the large cells is not so easy, and Bielschowsky has shown that the picture in tuberous sclerosis is not a unified one, but that both ganglion cells and glia cells must be sharply differentiated in these cases, the ganglion cells appearing only in the cortex and glia cells being greatly in excess. Some authors claim that the difficulty in differentiating the two types lies in the embryonic type of the large cells and is a proof of the congenital character of the process.

Following the consensus of opinion at present, Recklinghausen's neurofibromatosis and tuberous sclerosis are one and the same process caused by developmental anomalies in the spongiocytes (principal cells of Schwann's sheath and glia cells), but with different localization. Sclerotic plaques are frequently found in the cortex of Recklinghausen's disease in which large cells are predominant.

The author's case offers no clinical details as the patient was found in a comatose condition, and no previous history could be obtained. Macroscopically the meninges appeared normal; the left temporal region was swollen and the broadened convolutions were hard, leather-like to the touch and on section were pale yellowish-gray, little differentiated from the pure white of the medulla. The posterior portion of the convolution involved in the sclerotic process showed a pigeon-egg sized protuberance to which the pia is closely attached and which is a bright reddish-gray color on section.

Microscopic examination showed that the hardened portion consisted of an enormous development of glia fibers with here and there small wartlike projections containing great numbers of cells. Cells resembling normal glia cells were in the majority in the medullary areas—round nuclei almost without protoplasm. Other cells were seen with larger and paler, sometimes irregular, nuclei. Occasional cells showed much protoplasm, and their nuclei were usually laterally placed. The nervous tissue in these regions was severely injured in all its elements.

The small round tumor body seen in the posterior inferior portion of the affected region showed an entirely different picture and furnished the real subject of the author's article. Enormous cells—from 70 to 90 microns—were seen embedded in a network of fibers. The cells had long, branching processes which formed a network with fibers lying between the cells. Many cells had several nuclei which are near the border of the cell body and bound together by fine chromatin threads. The form and amount of chromatin in the nuclei vary greatly. Sometimes only one nucleus is present, but it is of great size, almost filling the cell. The more normal looking nuclei show a fine chromatin net and one or more well stained round nucleoli.

The author goes into great detail over the fibrillary content of many of these cells, describing many forms of cells with and without dendrites, etc., and concludes that a large number of the cells are ganglionary and that the small tumor is a glioganglio-blastoma of the brain. No Nissl's granula were observed in any of the tumor cells described.

The illustrations lack much in clearness of detail and in convincing power as to the correctness of the author's conclusions.

GURD, Ann Arbor, Mich.

CONTRIBUTION A' L'ETUDE DE L'ANATOMIE PATHOLOGIQUE DU DELIRE AIGU IDIOPATHIQUE (CONTRIBUTION TO THE STUDY OF THE PATHOLOGIC ANATOMY OF ACUTE IDIOPATHIC DELIRIUM). L. REDALIÉ, Schweiz. Arch. f. Neurol. u. Psychiat. **7**:35-48, 1920.

In reviewing the literature on this subject, the writer lays particular stress on the work of Ladame, whose contribution was abstracted in the March, 1920, issue of the ARCHIVES OF NEUROLOGY AND PSYCHIATRY, page 324.

The existence of an acute idiopathic delirium has been disputed by numerous writers, who view it not as a morbid entity, but as always symptomatic of some other malady. Redalié, however, believes, as does Ladame, that it is a morbid entity which is quite distinct from that of acute symptomatic delirium which occurs in the course of various infections and encephalitis. Ladame estimates its frequency as 0.25 per cent. of all psychoses.

The case reported in this connection was that of a woman, 43 years of age, who suddenly developed an acute psychosis, characterized by mental confusion, marked motor agitation, sitophobia and progressive general and cardiac weakness, which terminated in death after ten days. The illness followed a violent psychic shock based on the imprisonment of her husband. Necropsy revealed a small endothelioma attached to the dura mater in the region of the cerebellum. The blood vessels of the meninges were markedly injected and the subarachnoid spaces were edematous. The nerve cells of the cortex presented degenerative changes, without pronounced destruction, however; the nuclei were poor in chromatin. The neuroglia cells showed marked proliferation, particularly in the neighborhood of the blood vessels and about the prolongations of the pyramidal cells. There was marked neuronophagia. In the medullary substance, notably about the blood vessels, there was likewise an increase of glia cells. No changes of consequence were noted in the myelin or in the axis cylinders. Changes in the cerebellum were extensive and the description of these is essentially original with this writer. The granular layer was enormously changed by edema, the entire structure being transformed into a system of irregular cavities; the cells themselves contained pyknotic nuclei. Here and there, there seemed to be complete disappearance of the Purkinje cells; the cytoplasm of many stained an intense blue with toluidin, other cells were very pale and without nuclei or neucleoli. A scar, probably the result of an old ependymitis was found in the right lateral ventricle. In addition to these findings, there was epicardial ecchymosis and purulent bronchitis.

The cause and the mechanism by which these changes come about are unknown. Ladame thinks the changes are initially chemical in nature and due to lecithin disintegration; these, he thinks, may well be precipitated by a psychic trauma alone. Redalié thinks that infection has little to do with the disease, basing his belief for this assumption on the fact that there was complete absence of fever, absence of any history of preceding infection, of inflammatory foci in the brain, of hemorrhages or of evidence of transportation of disintegrated myelin; there was, on the other hand, a marked generalization of the process as the horn of Ammon was the only portion of the brain intact. The cause, he thinks, is an auto-intoxication, but he makes no attempt to formulate a more tangible hypothesis further than calling attention to the similarity of some of these findings to those described by Michailois in a case of Asiatic cholera.

The extensive damage done to the cerebellum was quite unexpected as there were no symptoms noted during the life of the patient that would direct attention to this organ. The only finding that might have been attributed to this was the absence of the patellar reflexes; it is possible that incoordination may have been present but if so, it was completely disguised by the extreme agitation of the patient.

WOLTMAN, Rochester, Minn.

LA POLYURIE HYPOPHYSAIRE (HYPOPHYSIAL POLYURIA).

E. SCHULMANN and R. DESOUTTER, Rev. de méd. **37**:441 (Sept.) 1920.

Under the name diabetes insipidus, or essential polyuria, one designates a syndrome characterized by an exaggerated and persistent emission of urine without glycosuria or albuminuria, and independent of any renal, cardiac or arterial disease.

The relation of diabetes insipidus and of hypophysial polyuria to each other is unsettled, though the frequent occurrence of anatomic hypophysial lesions in cases of diabetes insipidus is noted, and the author uses the term diabetes insipidus and hypophysial polyuria interchangeably. Is hypophysial polyuria a syndrome of pituitary hyperfunction, of hypofunction or of dysfunction?

Hypophysial polyuria may begin extremely suddenly. In some instances, the patient can recall the day or even the hour when the volume of urine augmented. An injury, a fright, or other strong emotion may, but need not necessarily, mark this day. In other cases, the onset is moderately rapid but spaced over one or two weeks, as a clinical development subsequent to an infection, a head injury, or parturition. Yet other cases are quite insidious and slow in development.

The urinary output in mild cases ranges from 8 to 15 liters in twenty-four hours upward to even 24 liters in that period in other recorded cases. The urine passed during the night period is in excess of that passed during the day. When a normal person and a patient with diabetes insipidus are tested regarding their response to the ingestion of a large amount of fluids, it is found that diuresis begins sooner in the normal. In the insipidus case, the augmentation of diuresis is more tardive but more durable.

The urine in these cases presents two characters. It is normal and it is dilute. The specific gravity is about 1.001 in the severe polyurias of 20 liters and possibly 1.004 to 1.006 in those of only 8 liters. Urinary chemistry does not reveal the presence of any pathologic element, provided there is no concomitant disease condition.

The writer is convinced that (apart from the coexistence of a renal lesion distinct in itself) the kidney in diabetes insipidus is not incapable of reaching a functional concentration of urine. This opinion seems justified since he has demonstrated a concentration of urine in persons with diabetes insipidus (1) in the course of febrile episodes, (2) in the course of partly successful attempts to suppress the ingestion of fluid, (3) under the influence of hypophysial medication, and (4) by adding to the habitual régime a salt, such as sodium chlorid or urea.

The arterial tension is neither exceptionally high nor particularly low and variations in urinary volume do not echo modifications of pressure.

The polyuria does not appear to be a function of blood pressure nor of an urea secretory constant. It is the result of a lowering of the kidney threshold for water. It is an exaggerated permeability of the kidney to water.

DAVIS, New York.

UEBER EINEN FALL VON ENCEPHALOMYELOMENINGITIS TYPHOSA (A CASE OF ENCEPHALOMYELOMENINGITIS TYPHOSA). HEINRICH AUGUST MÜLLER, Deutsch. Ztschr. f. Nervenh. **66**:168, 1920.

The fact is emphasized that abdominal typhoid fever frequently is associated with severe symptoms attributable to the central nervous system when anatomic lesions are not found of sufficient importance to explain the symptoms. Strümpell especially notes this fact in his textbook.

The author reviews the work of a great number of authors who, however, are far from furnishing a definite picture of the changes in the central nervous system caused by typhoid, and all the phenomena noted are seen in a variety of other diseases.

Probably the majority of observers today are of the opinion that the severe symptoms referable to the central nervous system are due to molecular injury of the nerve cells by the typhoid virus and not by the long continued elevation of temperature. Gerhardt is quoted as describing cases in which a lowering of temperature occurs concomitant with severe brain symptoms, and when the temperature rises these symptoms disappear. Gerloczy, Fräntzel and others are cited in favor of the toxic theory.

Many cases are also cited in which a meningitic syndrome was present, but an examination of the spinal fluid showed that a diagnosis of meningitis was incorrect.

On the other hand, many cases of meningitis are reported as occurring with typical abdominal typhoid, but the author's case differs from those already noted by the absence of intestinal localization and the presence of a typhoid meningitis. The diagnosis was made in life by finding typhoid bacilli in the spinal fluid which were placed in culture mediums with positive cultural results.

The clinical history of the author's case was briefly that of a man aged 48 years who became suddenly ill after eating a meat pie. He suffered from nausea and a feeling of heat, but continued in service for ten days. From the ninth day his temperature was taken regularly and reached 39 C. every evening with morning remissions. On the eighteenth day of his illness a careful examination showed pain in the occipital region with tenderness on pressure and much diminished knee jerks. Later he developed diplopia, bladder trouble and paresis of both legs with normal temperature.

There appeared progressive difference in pupils, slow reaction to light, paralysis of the right abducens and right facial nerves, also of the motor fifth and of the twelfth nerve, complete loss of patellar reflexes, etc. The patient's mind was perfectly clear during this period (from the twentieth to the twenty-third day of his illness). Neither typhus nor paratyphus bacilli were found in the blood, but Widal's reaction was positive, 1:1200. The paralytic symptoms increased in intensity up to a certain point, and sensory symptoms were present, but there was considerable variation in the severity of the paralysis up to the thirty-first day of his illness when his mind became unclear, he had difficulty in breathing and finally died.

Necropsy examination revealed small hemorrhages in all the internal organs, intestines included, but none of the ordinary lesions of typhoid in the intestines.

The brain showed some opacity of the pia-arachnoid but no alteration in the cranial nerves or basal vessels. The cortex showed extreme paleness of the white matter with hyperemia of the gray matter.

Microscopic examination showed small cell infiltration around the vessels and some small hemorrhages in the peduncles, in the oculomotorius nuclei, in

the locus ceruleus and in the hypoglossus area. The nerve cells in all these regions were practically unaltered. A few small softenings with large phagocytic cells were seen near the locus ceruleus. The manifestations were most severe in the peduncles and more severe in the medulla than in the pons. The spinal cord showed small cell infiltration about the vessels, severe hemorrhages and degeneration in the lateral columns. The horn cells were unaltered.

The pia arachnoid, particularly of the base of the brain, the medulla and the cord showed infiltration by leukocytes, especially around the vessels. The endothelial lining of the capillaries and the small vessels showed some swelling.

The author goes into the differential diagnosis at length, but probably the greatest value of the article lies in the full bibliography of articles on, and allied to, the subject under discussion.

GURD, Ann Arbor, Mich.

MEASLES: BRAIN COMPLICATIONS. A. L. SKOOG, J. A. M. A. **74**:1697 (June 19) 1920.

The author reports two cases in which both patients had brain complications following measles. One patient had a cerebellar syndrome, and made a complete recovery within six weeks. This case, the author believes, represents one in which the cerebellum and possibly the cerebellar tracts were involved directly from the virus of measles or its toxin. The second case was a complication involving the meninges and the cerebrum. Originally, this was probably a meningitis, appearing a few days after the onset of the measles, with some secondary septic organism as the cause.

From a neurologic review of the two cases reported and an analysis of the literature, the inference is drawn that measles are caused by some virus or organism as yet unseen and uncultivated; further, that the complications and sequelae involving the brain, spinal cord and peripheral nerves are uncommon. Still such cases do occur and may be divided into three groups. The first group would include a minor number of cases in which measles would be merely incidental in their relationship to the complication. The second group, by far the largest, would include complications due to a secondary invading organism. The third group, being a less certain one, would include conditions in which the exact etiology speaks for a direct invasion of the brain by the possible organism causing measles, or its toxin.

The prognosis depends entirely on the severity and location of the pathologic lesions in the brain. Very little is given relative to the treatment of these complications. In a final summary the author states that possibly a number of cases of obscure neurologic disorders, including those with a neurosthenic syndrome, might have had their origin in troubles involving the brain or meninges complicating measles or allied contagious diseases.

ADSON, Rochester, Minn.

THE IMPORTANCE OF VAGAL AND SPLANCHNIC AFFERENT IMPULSES ON THE ONSET AND COURSE OF TETANIA PARATHYROPRIVA. W. L. PALMER, Am. J. Physiol. **52**:581 (July) 1920.

This study was suggested by the frequent clinical association noted between tetany and gastro-intestinal disorder. Two methods of procedure were followed in these experiments. In the first, the effects of double vagotomy and splanchnectomy were studied on the course of tetany developing in a series of

sixteen dogs, following complete thyroparathyroidectomy. A three stage technic was followed in this group, that is, at the first operation the left splanchnic was destroyed, thoracically, and both vagi were cut; following this, at weekly intervals, the right splanchnic nerve and celiac ganglion were removed, and complete thyroparathyroidectomy was performed. In the second method of procedure, the effect of gastro-intestinal irritation (from 2 to 4 minims of croton oil daily) was determined on the tetany occurring in a series of fourteen thyroparathyroidectomized dogs.

The results obtained in this study were entirely negative. In the first series, 64 per cent. of the subjects developed tetany, a figure closely approximating the usual incidence of tetany in thyroparathyroidectomized animals, without vagotomy or splanchnectomy. In the second series, tetany developed in 78 per cent., but the disturbance was not so severe or prolonged as in the first group. It is of interest to note that the frequent vomiting observed by Carlson and Jacobson occurred in two animals of the first series, thus indicating a central origin for the emesis of tetany. The author concludes that neither vagotomy and splanchnectomy nor artificial gastro-intestinal irritation have any influence on the onset and course of tetania parathyropriva, and suggests the possibility of some chemical relationship in the association of gastro-intestinal disorder and tetany.

RAPHAEL, Kalamazoo, Mich.

LA SACRALISATION DE LA Ve LOMBAIRE (THE SACRALIZATION OF THE FIFTH LUMBAR VERTEBRA). L. DELHERN and THOYER-ROZAT, Bull. méd. **36**:6 (Jan. 1) 1921.

The authors call attention to an anatomic abnormality consisting of fusion, complete or incomplete, of the fifth lumbar vertebra, with the sacrum. This varies in degree from a relative enlargement of one or both transverse processes to a condition in which the body of the vertebra itself, owing to the great hypertrophy of a transverse process, is directly continuous with the sacrum and iliac bone.

The condition occasions pain symptoms explicable on a basis of root irritation, compression of surrounding tissue, ligamentous strain or possibly on a basis of an "arthritis" of the newly formed articulation between the hypertrophied process and sacrum or ilium. The diagnosis rests on radiographic discovery of the anomaly. Concomitant lesions must not be unsought, however. There is, especially, a tendency for the condition to be associated with spina bifida.

The writer believes that galvanism of the region, thermic penetration and particularly radiotherapy, are effective as treatment. In France, Nové-Josserand and Manclaire have practiced surgical removal of the enlarged processes.

DAVIS, New York.

BRAIN TUMOR AND TRAUMA. HUBSCHMANN, Deutsch. Ztschr. f. Nervenheilk. **66**:1 (May 26) 1920.

Hubschmann directs attention to the opportunity afforded by the war for the study of the relationship between head injuries and brain tumor. After giving brief histories and necropsy records in two cases of brain tumor in which a history of trauma was given, he reviews the findings of a number of other writers and gives the results in 107 brain tumor cases in which the

histories were accessible. In 17 per cent. of these cases there was a history of trauma, but in six of them either the injury was slight or head symptoms had been noted before the injury. In the remaining 11 per cent. there were a number in which the relationship between accident and tumor was doubtful. For instance, a 48-year-old man fell downstairs three months before his death. Three weeks later definite tumor symptoms were noted. Section showed a glioma. The accident here was doubtless due to an attack of dizziness caused by the already existing brain tumor. In only three cases were signs of a previous injury to be discovered at necropsy. G. B. Gruber is quoted to the effect that the latter found no case in his war material in which a blastoma could be said to be directly due to trauma, but the possibility of quickened growth, with manifestations of a heretofore existing occult glioma, is admitted.

INMAN, San Francisco.

A STUDY OF FORCED RESPIRATION; EXPERIMENTAL PRODUCTION OF TETANY. S. B. GRANT and A. GOLDMAN, Am. J. Physiol. **52**:209 (June) 1920.

Grant and Goldman report the consistent appearance of all the essential symptoms of tetany as a result of forced respiration in each of twenty-four experiments on two human subjects.

The subjects were required to breathe as deeply as possible, at the rate of fourteen inhalations a minute, while in the supine position, until symptoms of tetany developed, usually in from fifteen to sixty minutes. Blood and urine examinations were made, and the alveolar carbon dioxid tension measured, before and after each experiment. Diagnosis of tetany was based on the presence of carpopedal spasm, Chvostek's sign, Trousseau's sign, Erb's sign of increased electrical irritability and, in one instance, tetanic convulsion.

In addition to symptoms of tetany, there was found, uniformly, a fall of alveolar carbon dioxid tension which resulted in a reduction in hydrogen-ion concentration of the blood, a reduction of the carbon dioxid capacity of plasma, a change of urinary reaction to the alkaline side, a decreased excretion of ammonia and a slightly increased serum calcium content. The last finding is of especial interest as it stands in direct contrast to observations in clinical tetany made by other workers, notably by MacCallum and Voegtlin, MacCallum and Vogel, and Howland and Marriott.

In concluding, the authors suggest that tetany be regarded as a syndrome which probably may be caused by any condition tending to heighten the irritability of the peripheral nerves as, for example, in their study, the systemic alkalosis produced through forced respiration.

RAPHAEL, Kalamazoo, Mich.

NERVE SUTURE. EDWIN A. MILLER, M.D., Arch. Surg. **2**:167 (Jan.) 1921.

This experimental study was undertaken to ascertain the tensile strength of the suture line when enough of the nerve has been removed to require fixation in marked flexion of the limb. The conclusions are:

1. In dogs which show individual differences in rapidity of repair, as human beings do, the tensile strength of a suture line in the sciatic nerve or its branches is practically as great at the end of the third week as at the end of the fourth or fifth week.

2. The strength of the suture line, especially after the second week, is almost directly proportional to the diameter of the nerve.

3. The epineural sutures of fine catgut or silk play little, if any, part in the strength of the suture line, after the second week.

4. Long defects of nerves may be overcome by mobilization of the segments and posture, an end to end suture being performed. The suture line is apparently firm enough after three weeks to begin gradual straightening of the flexed forearm only. Clinically, after operation on the sciatic nerve, it would seem best to wait six or eight weeks after suture before extending the leg of flexion to complete an end to end suture. In case of the median and ulnar, extension of the forearm should not be attempted until after weeks of flexion.

RODMAN, Philadelphia.

LA ENCEFALITIS LETHARGICA EN ESPANA (LETHARGIC ENCEPHALITIS IN SPAIN). G. R. LAFORA, Arch. de Neurobiol. **1**: 209 (June) 1920.

This is a clinical and anatomopathologic study. Lafora summarizes the clinical and pathologic findings published, beginning with Economo and extending to the date of this article. He then presents several cases similar in clinical character and histopathology to those mentioned and goes into the theories of causation, etc. He offers nothing new, but he stresses the difference in the histopathologic picture of lethargic encephalitis (a more or less chronic inflammatory process with infiltration by lymphocytes and their derivates, plasma cells, etc.) and that of encephalitis occurring in influenza in which the lesions are purulent and hemorrhagic in type. He also notes the resemblance of the lesions in lethargic encephalitis to other brain lesions of parasitic origin, such as rabies, syphilis and sleeping sickness. He makes a point which he considers of importance, that is that every tissue has a characteristic reaction in relation to different germ groups, the brain for instance reacting to microbes (meningococcus, diplococcus, staphylococcus, etc.) by polymorphonuclears thus giving rise to acute meningitis or to abscesses in the substance of the brain, on the contrary reacting to parasites by lymphocytic focal or diffuse infiltrations of the blood vessels and meninges, Koch's bacillus alone furnishing a parasitic reaction.

GURD, Ann Arbor, Mich.

POLYNEURITIS FOLLOWING INJURIES. FRIEDRICH LEPPMANN, Ztschr. f. die ges. Neurol. u. Psychiat. **49**: 198 (July 11) 1919.

Leppmann takes up the question as to whether widespread nerve inflammation as a sequence of trauma can occur in the absence of external injury and suppuration. In addition to several doubtful cases cited from the literature, he gives the history of one case observed by him in which the element of accident compensation came into question. However, the possibility of a poliomyelitis or of the effect of overindulgence in alcohol could not be excluded. Numerous brief case histories are cited in which a more or less widespread neuritis followed infected wounds.

In conclusion Leppmann states that there is no evidence in proof of a widespread neuritis resulting from uninfected wounds, and that a polyneuritis following trauma without tissue laceration or infection will be found to be due to some other cause, such as diphtheria.

INMAN, San Francisco.

A PLETHYSMOGRAPHIC STUDY OF SHOCK AND STAMMERING IN A TREPHINED STAMMERER. S. D. Robbins, Am. J. Physiol. **52**:168 (May) 1920.

The subject of this plethysmographic study was a man, aged 45, who, as a result of operative interference, fifteen years previously, following a bullet wound of the head, was left with a slightly depressed trephine opening, about 2.5 cm. in diameter, in the right frontotemporal region, 5 cm. from the midline. There was a history of stammering since early childhood, following fright but, at the time of the experiment, speech was practically without defec. when the subject was alone.

On the basis of this study, the author reports marked increase in brain volume, in association with stammering, as compared with the lesser increase determined in the course of normal speech, thus corroborating Bluemel's hypothesis. Robbins also noted increased brain volume in association with strong emotional ("shock") reaction and mental and physical work, bearing out earlier work by other observers, notably by Berger, Mosso, Shepard and Weber.

Raphael, Kalamazoo, Mich.

VARIATION DU TAUX DE L'UREE DANS LE LIQUIDE CEPHALO-RACHIDIEN PRELEVE AU MOMENT ET EN DEHORS DES CRISES CONVULSIVES EPILEPTIQUES ET HYSTERIQUES (VARIATIONS IN THE UREA CONTENT OF THE CEREBROSPINAL FLUID OF EPILEPTIC AND HYSTERIC PATIENTS, TAKEN DURING CONVULSIVE SEIZURES AND AT OTHER TIMES). Gaston Laurès and Emile Gascard, Presse méd. **28**:396 (June 16) 1920.

"Whatever the urea content may be at other times, it is lessened during the hysterical and increased during the epileptic convulsion." To support this thesis, the findings in six frankly epileptic and six hysterical cases are cited; also in two doubtful cases, which yielded results of epileptic type and later developed unquestioned clinical epilepsy. Uremic and syphilitic convulsions, if definitely epileptiform, were not positively differentiated from idiopathic grand mal, and the fluids of such cases conformed to the epileptic type.

Lumbar puncture was performed during a postconvulsive phase, and again four days later, in each case studied. The following figures represent grams of urea in 1,000 c.c. of cerebrospinal fluid: average epileptic content during convulsion, 0.55; otherwise, 0.33; during hysterical convulsion, 0.28; otherwise, 0.47.

Huddleson, New York.

DISEASE OF THE PERIPHERAL NERVES IN THE WAR. Erwin Wexberg, Ztschr. f. d. ges. Neurol. u. Psychiat. **49**:198 (July 11) 1919.

In this article of seventy-one pages, the author presents case histories and discusses the probable causes of the peripheral nerve disturbances occurring in the Maria Theresa Schossel in Vienna. The cases fell into five groups. In nine of the fifteen cases in the idiopathic polyneuritis group, exposure to cold had been given as the causative factor. However, in only one case did this cause alone seem to be culpable. In three other patients there had been associated nonspecific gastro-intestinal disturbances, while the remainder had suffered from undernourishment, which was assumed to have reduced their resistance.

Inman, San Francisco.

SOBRE LA COMPOSICION Y LAS PROPRIEDADES DEL LIQUIDO CEFALORAQUIDEO EN LA ENCEFALITIS LETARGICA (A STUDY OF THE COMPOSITION AND PROPERTIES OF THE CEREBROSPINAL FLUID IN LETHARGIC ENCEPHALITIS). W. MESTREZAT and BELARMINO RODRIGUEZ, Arch. de Neurobiol. **1**:227 (June) 1920.

Four cases are studied and the authors conclude that in lethargic encephalitis, with the exception of hyperalbuminosis and hypercytosis, the cerebrospinal fluid is essentially and generally normal and its chemical, cytologic and serologic examination offers an important diagnostic method in this disease, which is so variable in its clinical manifestations.

GURD, Ann Arbor, Mich.

Society Transactions

THE CHICAGO OPHTHALMOLOGICAL AND THE CHICAGO NEUROLOGICAL SOCIETIES

Joint Meeting, Dec. 16, 1920

ALFRED N. MURRAY, M.D., *President of the Chicago Ophthalmological Society, in the Chair*

A CASE OF POLIENCEPHALITIS SUPERIOR AND INFERIOR. DR. G. B. HASSIN.

This article is published in full in this issue, p. 552.

DISCUSSION

DR. HIRAM J. SMITH said that the cause of ocular paralysis may be orbital or intracranial. The intracranial may be considered as supranuclear, nuclear fascicular, or superficial. In extensive progressive ophthalmoplegia the lesion is nearly always nuclear. Supranuclear lesions, that is, of cortex association centers and intracerebral tracts, cause conjugate paralyses, seldom isolated paralysis, with the exception of ptosis. In this type of conjugate paralysis, the eyes usually are able to turn toward the affected side of the brain, but not toward the opposite, that is, "look to the lesion," as contrasted with conjugate paralysis of pontile origin, in which the eyes might turn from the lesion.

Bilateral ophthalmoplegia is not necessarily due to bilateral involvement of the nuclei. Fibers from nuclei of a given side pass to the nuclei of the opposite side so that a lesion of the right third nucleus may cause a disturbance of the muscles of the opposite side, as ptosis. The affection of the opposite side, in a case under observation, cleared up in forty-eight hours, probably through compensatory action of the unaffected nucleus, as the paralysis on the side of the lesion persisted.

In progressive nuclear involvement, one would naturally expect the adjacent nuclei to be affected at the same time, or in succession; and this happened. The third and fourth, or sixth and seventh, were involved together, as well as adjacent nuclei of other cranial nerves.

The diagnosis of the cause of ocular paralysis might be suggested by the type and extent of the trouble. In nuclear paralysis the underlying affection would be cleared up, usually through the finding of other manifestations than the ocular. The characteristics of multiple sclerosis were readily perceived. In bulbar paralysis the early involvement of hypoglossus and glossopharyngeus was encountered. In myasthenia gravis double ptosis was seen early, but rapid fatigue of the muscles of the head and neck, especially muscles of mastication, was characteristic. Nevertheless, many obscure clinical pictures presented themselves.

DR. PETER BASSOE thought it would be profitable if Dr. Hassin would emphasize the distinction between poliencephalitis superior and inferior caused by other infections and the purely degenerative affections of the same regions. A similar problem had been worked out in the case of the spinal cord. For a

long time all conditions were called myelitis without sufficient distinction between inflammation, degeneration and vascular lesions.

Dr. H. Douglas Singer stated that, according to the statement of the essayist, the spinal fluid was absorbed apparently both through the arachnoid villi and through the choroid plexus. He wondered what the source of the spinal fluid was and whether it was true that the fluid was absorbed at both sides of the brain. The usual view was that the choroid plexus acted as a gland to create the fluid, but Dr. Hassin apparently had an altogether different view.

Dr. Michael Goldenburg said that he was under the impression that the spinal fluid was secreted by the choroid plexus, and that the epithelium covering it was merely a filter.

Dr. Hugh T. Patrick asked how the fat and epithelial cells in the choroid plexus got there from the spinal fluid, and whether anybody had ever found fat in the spinal fluid in this sort of case.

Dr. Hassin, in closing the discussion, said that he did not intend to consider in detail the important physiologic points suggested by the pathologic studies of a remarkable case. He merely wished to demonstrate their probable significance. The masses of lipoid substances in the gray matter of the midbrain and medulla were striking, but nobody had ever noted their presence in the subarachnoid space and the choroid plexus. Evidently, fatlike substances had not been looked for in these regions or proper methods were not used. In fact, few histopathologic studies of so-called hemorrhagic superior poliencephalitis had been recorded, the authors contenting themselves with repeating what Wernicke had said. Schroeder and Spielmeyer were the first to point out that Wernicke's poliencephalitis was not an encephalitis at all. In his (Dr. Hassin's) opinion the only true superior poliencephalitis was represented by epidemic (lethargic) encephalitis in which the inflammatory phenomena were principally, though not exclusively, confined to the midbrain. In Wernicke's type the morbid process had the same localization, but was of a degenerative, and not of inflammatory, character.

As to the probable function of the choroid plexus, he wished to state that, according to some authors, the cerebrospinal fluid originates partly in the brain, partly in the choroid plexus. The abundance of fat in both these structures indicated that their contents were wholly derived from the brain tissues. In the case under discussion, these contents were lipoid substances; in cerebral hemorrhages they would be blood pigment and so forth. The choroid plexus, therefore, was to be looked on as a filter for the cerebrospinal fluid, which it rendered passable through the various channels of absorption. Generally speaking, the study of pathologic brain conditions might help to solve problems which so far defied the efforts of the ablest experimental workers.

THE PUPIL IN HEALTH. Dr. E. V. L. Brown.

Dr. Brown stated that, according to Salzmann, the pupil in health had a diameter of approximately 4 mm. The consensual reaction depended on the stimulation of the rods and cones in the relatively small area of the macula. The stimulus was then carried by the optic nerve to the chiasm, where partial decussation took place, thence via the tractus opticus with the pupillary fibers lying dorsolateral to the corpora quadrigemina, and finally to the nucleus of the oculomotor nerve, which functioned as the pupil nucleus as well. Through

the fibers which crossed over from the right to the left side, therefore, any stimulus of the right macula went to the left pupil, centered as well as to the right and was then sent down the left oculomotor to the sphincter of the iris on each side, the left pupil narrowing at the same time the right did. This test was of the greatest value in establishing the functional integrity of the most vital part of any injured eye. Many a patient who had just suffered a severe accident to a considerable portion of the front of his eye could easily and quickly and honestly be told that the eye was not lost, even when the cornea was cut, the anterior chamber full of blood, the iris prolapsed or the lens dislocated, for the pupil of the fellow eye narrowed when light was thrown into the injured eye. One of the uncanny things about the consensual pupil reaction was the fact that in rare instances of disease of the cortex, such as tumor of the occipital lobe, etc., one eye actually did not see, or rather the cortex of neither side saw, yet the pupil motor stimulus was sent up the one optic nerve, across to the other side and down that oculomotor nerve, and the pupil of the opposite side narrowed as perfectly as if light and color and form perception were perfect.

Widening of the pupil takes place through irritation of the sympathetic nerve. The pupil widening fibers leave the spinal cord at the level of the upper two dorsal and the lower cervical segments. Fibers from the upper thoracic ganglion and the anterior branch of the ansa Vieussenii go to the inferior cervical ganglion, and out of it into the cervical ganglion via the cervical sympathetic. Here there is union with the hypoglossal. The carotid branches are then given off and the pupillodilatator fibers proceed in the skull to the Gasserian ganglion and unite with the first branch of the trigeminus. So united they proceed to the eye via two long ciliary nerves to the dilator sheet of muscle in the back layers of the iris. They do not pass through the ciliary ganglion at all.

Any irritation of the cervical sympathetic can, therefore, produce dilatation of the pupil. Furthermore, the irritation or stimulation of any sensory nerve may produce a dilatation of the pupil. The path here is to the cerebral cortex, the oculomotor nucleus and to the iris via the third nerve, ciliary ganglion and short ciliary nerves to the sphincter pupillae, which relaxes and allows the dilatator to work unopposed. Furthermore, the pupil widens on any psychic stimulus, and volitional impulse and any vivid mental concept.

DISCUSSION

Dr H. Douglas Singer stated he often found recorded: "pupils sluggish to light," and he had never been able to satisfy himself as to what most people meant by sluggishness. Did it mean that the reaction was slow or that the degree of contraction was diminished?

In his opinion the fibers that conveyed the stimulus for the light reflex left the optic tract before it reached the pulvinar. They apparently left in the region of the thalamus and traveled along the inner side of the thalamus. This seemed to be proved by two cases of tumors seen many years ago, involving the back part of the third ventricle and damaging the optic thalamus on both sides in both of which there had been Argyll Robertson pupils.

Dr. Hugh T. Patrick stated that the dictum of Uthoff that even if there was more illumination of the pupil on one side, the pupils remained equal, was wrong.

Another curiosity could be referred to as a normal pupil: It was known that occasionally a person could voluntarily dilate his pupil by picturing to himself some peculiarly horrible scene, generally from his own experience.

Dr. Ralph C. Hamill said that in testing the pupils, especially of colored men with dark irides, it was difficult to tell whether there was a light reflex or not. Dr. Brown had mentioned the fact that the near-sighted person has small pupils, and he wondered whether in some persons where the pupil was under more or less spasm small changes of size would be visible. Also, in testing the pupils of a great many men in a short space of time, as was done with some of the men in the training camps, it was observed that there were certain kinds of pupils that corresponded to the degree of pigmentation of the iris.

Dr. I. Leon Meyers thought that the influence of the sympathetic nervous system, and especially that which was noted in emotional disturbances, fright, etc., was not the only one that brought about dilatation of the pupil. It had been noted many years ago that in stimulating the cortex of an animal while it was completely anesthetized and the stimulation was strong enough to produce epileptiform fits, the pupils would promptly dilate. This had no connection with stimulation of the cortex when it produced conjugate deviation of the head and eyes.

Dr. Robert von der Heydt stated that as to light and dark irides; there were at least two reasons why eyes with dark irides did not respond to light as well as those with lighter colored irides. One was a sluggishness in response on account of the weight of the added pigmentation in dark irides. Then, light would penetrate a light colored iris more readily on account of its greater transparency, and the retina would receive more stimulation for that reason.

Dr. H. W. Woodruff spoke of the statement made in the standard textbooks on ophthalmology that "inequality of the pupils was always pathological." Reference had already been made to the larger pupil in myopia. This also held when one pupil was myopic and one hyperatrophic, namely, in antisomatropia. In such a case one pupil was distinctly larger than the other. When he first began the practice of ophthalmology he did not know this and supposed a patient with inequality in the pupils must have a serious nerve lesion. For this reason, in examining these cases, the refraction should be known.

Dr. Charles P. Small said that the differences in the reactions in the normal pupil were illustrated in a case seen recently. The patient was a man in perfect health, with all laboratory examinations negative, who was refused an increase in life insurance because he was said to have an Argyll Robertson pupil. The pupils were widely dilated and almost immobile, but they did react sluggishly when carefully examined. He did not know why he had such feeble reaction, and wished some of the neurologists would explain it to him.

Dr. C. W. Hawley was reminded of a case similar to Dr. Small's which he had reported. His patient had widely dilated pupils all her life without pupillary reaction. Suddenly the left pupil was contracted to the usual size and developed reaction. She came to have the pupil dilated to look like the other.

As to one pupil dilating more when it was receiving more light than the other, he had seen a similar case within two or three months. During the examination a friend of the patient asked why one pupil was dilated more than the other, and he thought it might be because that eye was receiving more

light than the other. On turning the patient around he obtained the opposite effect and proved that this theory was correct.

Dr. Brown, in closing the discussion, in reply to Dr. Singer, said he had always understood sluggishness to refer to the rate of reaction rather than the degree. He was glad to hear Dr. Patrick emphasize the fact that when more light enters one eye than the other the pupil of the first eye is narrower than that of the other, thus proving that the direct reaction is greater than the indirect or consensual reaction.

PHILADELPHIA NEUROLOGICAL SOCIETY

Jan. 28, 1921

Charles S. Potts, M.D., *President pro tem.*

A CASE OF PARALYSIS WITH CONTRACTURE OF PHYSIOPATHIC ORIGIN. Presented by Dr. A. J. Ostheimer.

An ex-service man, now 20 years of age, fell into a shell hole while carrying a wounded soldier back from the line on July 18, 1918. The result, according to a report from the Adjutant-General's office, was a simple fracture of the body of the left scapula, with dislocation of the left humerus, causing injury to the third, fourth and fifth cervical nerves. A roentgenogram made at the time of discharge, April 9, 1920, showed separation of the glenoid cavity, with some atrophy of the infraspinatus, supraspinatus and deltoid.

When examined, June 29, 1920, there was a good deal of dragging pain in the left shoulder, along the trapezius and in the hand, which at times became somewhat contracted in a flexed position. Both hands and feet were of a somewhat dusky hue, mottled and showed profuse perspiration. He was unable to raise the left arm to the horizontal. There was involvement of the left trapezius, levator scapulae, rhomboidei and to a much lesser extent of the serratus magnus and supraspinatus. As these muscles are supplied chiefly by the third, fourth and fifth cervical nerves, there seemed to have been a rupture of the cervical plexus. The left grip was 35 on the dynamometer, the right 85; otherwise there was no evidence of organic trouble.

A roentgenogram made on July 9, 1920, showed no abnormality. About this time the patient began gradually to develop marked contracture of the left forearm and of the hand on the forearm with paralysis, together with the thermic, trophic and vasomotor symptoms which accompany the reflex disorders, as Babinski and Froment have named them, or the dynamoneuropathic disorders under which name the late E. E. Southard has classified them. All the typical signs of the "main figeé," as first described by Meige, were present. The vasomotor disturbances were marked, confined to the hand and did not correspond to any innervation area. The right hand also was cold and showed slight global atrophy, while the skin was of salmon color, wet and mottled. The mechanical excitability of the small muscles of the hand, particularly of the thenar and hypothenar eminences, was increased, as well as the electrical excitability of the same muscles. All the other muscles showed normal electrical reactions. The tendon reflexes were practically normal. The paralysis was almost complete. There were no sensory disturbances.

This case is of interest because the symptoms and signs are different from those that occur in frank lesions of the nerves, as well as from those that are purely pithiatic. The thermic, trophic and vasomotor signs, as well as the resistance to all forms of treatment, makes this condition a distinct entity. The recent splitting of functional cases into the psychopathic and the physiopathic has been well worked out, principally by Babinski and his associates of the French school. Hurst and other English neurologists have held that all of these symptoms are simply consequent on disuse following pithiatic paralysis and contracture. By chloroforming a patient, Babinski has shown that when all the other reflexes were stilled, certain reflexes that were entirely concealed in the waking state were brought out; yet at the same time consciousness in the usual sense of that term had vanished. Was not this proof of a new type of functional disease nonpsychic in nature, but of almost equally complex nature? The chloroform apparently suspended the operation of numerous neurons that have to do with the down flow of cerebral inhibitions and allows phenomena to appear in certain reflex arcs that argue an excess of activity, for example an ankle-clonus or a patellar clonus. This seemed to prove the existence of a disorder of a reflex or physiopathic nature below the level of the psyche and below the theater of operations of hysteria.

The most complete recent review of this subject has been made by A. Pitres and A. Laffaille: "Sur les paralysies globales molles fonctionnelles de la main consecutive à des blessures de guerre," in the *Revue de médicine*, 1920, Nos. 5 and 7.

DISCUSSION

Dr. William G. Spiller said this subject is important and has caused much discussion. Roussy in his paper read before the American Neurological Association last spring and in his moving pictures showed that the lesions of "reflex" contracture and paralysis are dependent on functional disturbance. He showed in moving pictures that when a paralyzed hand is moved synchronously with the sound hand, although at first the movement may be slight, the paralyzed hand slowly acquires power, and with the return of voluntary motion the vasomotor and trophic symptoms disappear. The functional character of the paralysis is demonstrated by the improvement under the treatment employed. The hysterical palsy thus seems to condition the vasomotor and trophic disturbances which in reality are organic but secondary to functional disturbances.

Dr. T. H. Weisenburg said that he had seen a number of cases similar to the one reported by Dr. Ostheimer, and he had no doubt that this case was purely functional. In a similar case, which had come under his observation in Plattsburg, by bullying the patient, in about five minutes he was able to extend his fingers whereas previously they were flexed. Subsequently this man was cured. One of the interesting points about all of these functional cases is the rapid disappearance of vasomotor phenomena not only in the case as described by Dr. Ostheimer, but in all cases of hysterical weakness and contracture.

Dr. D. Nathan said that abroad he had seen many cases very much like this in which there were contracture of the biceps tendon and atrophy and contracture of the hand. These cases were often associated with unilateral sweating and other vasomotor phenomena, and he classed them all as hysterical, and cured most of the patients in a few hours or days.

SYPHILIS OF THE EIGHTH NERVE. Presented by Dr. J. Hendrie Lloyd.

Dr. Lloyd based his paper on a series of cases observed in his hospital service. Sudden or rapid deafness, with tinnitus, coming on usually early in the secondary stage and ending in incurable loss of hearing, was the chief characteristic. In some cases there was a cranial polyneuritis, the seventh, fifth, third and second nerves being involved in about the order named. Severe headache with high lymphocytosis pointed to an acute basilar syphilitic meningitis as the cause. An opportunity for postmortem study occurred in one case, and the microscope recorded extensive destruction of the eighth nerve. The paper will be published in full in a later issue of the Archives.

DISCUSSION

Dr. N. W. Winkelman said that he had not yet completed the pathologic examination of the specimen referred to by Dr. Lloyd. The nerve was rather peculiar on both sides. It showed practically complete degeneration, only a few waxy axis cylinders running through. The neuroglia tissue was laid down in columns showing degeneration had taken place slowly. There were some concentrically arranged bodies that were probably myelin bodies in the hematoxylin-eosin and phosphotungstic acid stain, also some infiltrating mononuclear cells very hard to distinguish from the resting stage of the glia cells.

Dr. T. H. Weisenburg said that he did not agree with Dr. Lloyd that arsenic preparations had injurious effects on the auditory nerve or on any portion of the nervous system, and in his opinion they were distinctly the best antisyphilitic drugs we possessed. In the Dermatological Laboratories in Philadelphia the weekly output is over 10,000 tubes, and this laboratory is only one among many. Arsenic has been used for about fifteen years, a sufficient time for us to come to a conclusion regarding its influence on the nervous system, and up to the present there is no evidence of its deleterious effect.

Dr. William G. Spiller said that often there is meningitis in tabes, though sometimes slight, but it is not proper to attribute the degeneration of the posterior roots to the meningitis. Syphilis may by its toxic properties directly affect the posterior roots. He felt that sometimes too sharp localizations are made by the Bárány tests, because only one group of fibers in the acoustic nerve seems from the symptoms to be diseased, and the conclusion is formed that the lesion must therefore be where the fibers of this nerve having different functions have separated. This conclusion is unreliable. If one examines the lumbar region in a tabetic cord when death has occurred from some other disease, he will find the degeneration of the posterior columns corresponds to the embryologic picture afforded by the medullation of these columns at a definite period. The syphilitic toxin has attacked the whole of the posterior root, but only certain fibers in this root corresponding to their period of embryological "ripening" have succumbed, although later other fibers in the root would also have degenerated.

There are many examples of the selective action of poisons or pressure on certain groups of fibers in a bundle of nerve fibers. The papulomacular bundle is especially liable to degeneration from alcohol, lead or nicotin, also from pressure on the optic nerve. It alone of the optic nerve fibers may be affected in some cases of multiple sclerosis. Diphtheria may select the branch of the oculomotor nerve to the ciliary muscle without affecting the remainder of

the oculomotor nerve. Lead may select the posterior interosseous fibers from the musculospiral nerve. Therefore, because only the cochlear fibers or the vestibular fibers of the acoustic nerve have caused symptoms, it may be a mistake to assert that the lesion can by no means be within or on the acoustic nerve but must be within the brain stem where the tracts are widely separated. It may be at such a part but it need not be. Gowers has shown that clinically all of the fibers of the acoustic nerve are not invariably equally affected in tabes.

Dr. J. Hendrie Lloyd, in closing, said that in the main he agreed that arsphenamin is given so much with so little bad effects, that it is not fair to ascribe to this drug injurious effects on the auditory nerve. Nevertheless, there may be a weak spot in that argument, for it is the exceptional case that we must bear in mind. He was averse to making sensational statements, but he thought we ought to know the truth.

With reference to the Bárány tests, as he said in his paper, we should be slow in accepting some of the extreme claims made for their localizing value. They apparently have a general value in these cases of peripheral, or eighth nerve lesions, as indicating that some at least of the functions of the vestibular nerve are affected. The microscopic examination shows extensive destruction of the eighth nerve, and to that extent the Bárány tests were accurate in their results. This opportunity to examine the eighth nerve in acute syphilis was a rare one.

A CASE OF RAYNAUD'S DISEASE MUCH IMPROVED BY BRACHIAL SYMPATHECTOMY. Presented by Drs. George Muller and George Wilson.

The case shown was that of a man 72 years of age who had had symptoms and signs of Raynaud's disease in both hands for two winters. Following bilateral brachial sympathectomy there was marked improvement in the vasomotor phenomena in the hands.

The case will be presented in full at a later date.

FIVE CASES OF PITUITARY DISEASE. Presented by Drs. T. H. Weisenburg, C. A. Patten and F. Ahlfeldt.

Case 1.—*History.*—Z. S. H., a white woman, aged 35, whose family history was negative, except for the possibility of syphilis, had a normal birth. She was fat as a baby and delayed walking until she was 4 years old; she did not talk until she was 5. She had a persistent low mentality. The menses were established at 11 years; she had had nine pregnancies with five miscarriages and four stillbirths. An ankle was fractured ten years ago. She has had tremors of the left arm since being struck by her husband four years ago. Her hair became gray at the age of 11.

Physical Examination.—She had disproportionately long extremities; fat, small, pudgy hands; segmental distribution of the body fat, and a low hairline on the forehead. She had an adrenal "white line"; normal blood pressure, and a positive oculocardiac reflex. There were reaction to epinephrin with lowering of pulse (2 beats), increase of blood pressure (10 mm.), and a leukocytic increase from 8,600 to 10,800, a relative lymphocytosis and eosinophilia. She reacted to atropin with a rise of 20 beats in the pulse rate. The

sella turcica was distorted. She had a markedly increased sugar tolerance, and the blood Wassermann reaction was positive.

This case is interpreted as persistent hypofunction of the pituitary gland. In addition, the patient had a neurosis and was syphilitic.

CASE 2.—*History.*—M. F. was a white woman, aged 40. There had been endocrine disturbances in various members of the family: giantism, obesity, dwarfism, bed-wetting, diabetes and gastro-intestinal disorders. At the age of 5 she changed from a bright and active child to a dull and inactive one. Secondary sexual characteristics appeared at the age of 5, and a sudden increase of growth and weight at 9 coincident with the establishment of the menses. Bed-wetting had been present all her life. Two illegitimate pregnancies followed the awakening of sexual desires at the age of 18. Surgical menopause occurred at 26, with convulsive seizures occurring at intervals since, and coming on about the time of the usual monthly period.

Physical Examination.—The pupils were irregular. "Vessels in the left eye have a turn toward the nasal side on emerging from the nerve head." The body was disproportionately longer than the extremities. She weighed 330 pounds, with segmental distribution of fat. The hairline was low on the forehead. There were an adrenal "white line," low blood pressure and pulse rate. The oculocardiac reflex was positive. There was a reaction to epinephrin with an increase in the pulse rate (10 beats) and blood pressure (15 mm.); a leukocytic increase of 3,800 cells occurred—the lymphocytes and eosinophils were mainly affeted. There was a reaction to atropin with an increase in pulse rate of 20 beats a minute. The sella turcica was enlarged. There were increased sugar tolerance, lessened kidney elimination and a positive blood Wassermann reaction.

This case shows definite pituitary deficiency together with thyroid deficiency and artificial menopause. The patient was also epileptic (?) and syphilitic.

CASE 3.—*History.*—The case of F. F., a white girl aged 14, was fully reported by Dr. F. H. Leavitt before the Philadelphia Neurological Society on Dec. 19, 1919, and appeared in the ARCHIVES OF NEUROLOGY AND PSYCHIATRY, April, 1920, on page 452. It is included in this series for contrast.

Her family history was negative. Her birth was instrumental. She had a probable meningitis at the age of 8 months, marked by retraction of the head, convulsions, incontinence, and blindness (which lasted until the age of 2). Walking and talking were delayed. Her mentality had always been low. At 5 she became very fat and weak with evidence of hydrocephalus and lateral column sclerosis. Secondary sexual characteristics appeared at the age of 7 and menses at 12.

Physical Examination.—Examination revealed: enlarged head (60 cm.); small infantile extremities and a disproportionately longer torso; dry, doughy skin; hypotrichosis, and low hairline on the forehead. Recently her hair had been falling out. There was segmental distribution of fat and lymphoid tissue hyperplasia. The adrenal "white line" was present. There were a positive oculocardiac reflex, markedly increased sugar tolerance and an enlarged sella turcica. Reaction to atropin was strongly positive . Neurologically she showed a lateral column sclerosis and divergent strabismus. The blood Wassermann reaction was positive.

This patient revealed a definite hypopituitarism, probably secondary to the meningitis at 8 months and developing coincidently with the hydrocephalus and lateral sclerosis. She was also syphilitiic.

CASE 4.—*History.*—C. T., a colored boy, aged 15, with a negative family history, was normal up to 6 years of age when the right arm and hand began to grow disproportionately larger. At the age of 12 it was discovered that he had a positive blood Wassermann reaction. Antisyphilitic treatment was continued for one year. At 14, he began having convulsions described as typical petit mal and grand mal attacks. For the past few months these attacks have been more frequent.

Physical Examination.—This revealed: disproportionate length and circumference of the right arm, hand and shoulder and disproportionately short torso. The extremities showed proportionately longer proximal than distal length. The sella turcica was normal. There was a slight tufting of the distal phalanges of the fingers. The upper teeth were widely spaced; the canines were not fang-shaped, but very like the incisors. There were: moderately increased sugar tolerance, high lymphocyte count, low kidney function and increased nonprotein nitrogen in the blood. The Wassermann reaction was weakly positive to negative.

This case offers more difficulty in diagnosis. The evidence may possibly point to a hyperfunction of the anterior lobe in childhood, but it seems to have become arrested. At present he cannot be said to have hypopituitarism on the basis of a slightly increased sugar tolerance, except it be that this is evidence of the beginning of deficient function. It would probably be best to classify the case as one of dyspituitarism. The patient was also epileptic and syphilitic.

CASE 5.—*History.*—I. E., a white boy, aged 16, began developing rapidly at the age of 9. His father and mother were syphilitic and were first cousins. The father contracted syphilis at the age of 20, and the patient was the first born. The mother had been pregnant seven times with two miscarriages. There were three sisters and one brother—all showing endocrine disturbances, mainly in precocious physical and sexual development. The girls began menstruating at the age of 8 or 9 years, and their secondary physical characteristics appeared at the same time; the boy, now 16, had fully developed secondary sexual characteristics, axillary and pubic hair, adult genitalia and deep voice.

The patient had "snuffles" at birth. Walking and talking were delayed. He had had otitis media and interstitial keratitis in infancy and scarlet fever following diphtheria at 11, during which illness he began to grow rapidly and to gain in weight. Bed-wetting began also at this time and has continued since. Pubertal changes occurred at 13, but he has no sexual desires. Adult stature was reached at 13.

Physical Examination.—This revealed: partial loss of vision and impaired hearing; marked obesity (he weighed about 230 pounds), with a segmental distribution of the fat; disproportionately long extremities, especially the sacral portions; thin skin but doughy in consistency and hypotrichosis with low hairline on the forehead; about normal pulse and blood pressure. An "adrenal white line"; negative oculocardiac reflex; markedly increased sugar tolerance; low kidney elimination and positive blood Wassermann reaction; reaction to epinephrin with increase of pulse (18 beats) and increase of blood pressure (20 mm.); leukocytosis of 10,000 to 12,000; a positive reaction to atropin (pulse rate increasing 22 beats) and pilocarpin (decreased 4 beats). The roentgen ray showed an enlarged sella turcica and the convolutional markings of internal hydrocephalus.

This case evidently is one of dyspituitarism; the condition now is that of hypofunction, but evidence points to an earlier hyperactivity, at least of the pars anterior. He also has internal hydrocephalus, optic atrophy, bilateral impairment of hearing, enuresis and syphilis.

Remarks.—A positive blood Wassermann reaction is now, or has been, present in all five cases. The rôle which syphilis has played in the etiology of the dysfunction of the gland is perhaps not clear, yet the coincidence is striking. In Case 5 there is no doubt about the heredity of the disease and Case 4 is quitè certainly congenital; in Cases 2 and 1, syphilis was undoubtedly acquired, but in Case 3 the parents are definitely nonsyphilitic and the patient has never been exposed except it be through extragenital routes. There is definite evidence of endocrine disturbances in the families of Cases 3 and 5. Cases 3 and 5 give evidence of hydrocephalus and organic disease of the nervous system, while Case 1 shows a functional condition. All patients are of low mentality except the one in Case 4. Of the patients demonstrating hypofunction, bed-wetting is present in three; segmental distribution of fat is present in all, a low hairline on the forehead, an adrenal "white line," eosinophilia and lymphocytosis, disproportionate torso-leg ratio, changes in the sella turcica, crowding of the teeth on the lower jaw, markedly increased sugar tolerance, low kidney elimination and disturbed metabolism, are likewise present in all. In minor respects the cases, of course, differ somewhat, but these changes perhaps are dependent more on the disturbances of the interrelated endocrine organs and the underlying physical conditions. Particular attention has been given to the pituitary disturbances alone. The investigation of the other internal secretory organs has been left for further study; they have been mentioned here only in so far as they have a direct bearing on the interpretation of the findings.

NEW YORK NEUROLOGICAL SOCIETY

Three Hundred and Eighty-Sixth Regular Meeting

FOSTER KENNEDY, M.D., *President, in the Chair*

PRESENTATION OF CASE OF INFANTILE NUCLEAR APLASIA OR INFANTILE MOTOR DEFECTS IN THE CRANIAL NERVES.

DR. I. ABRAHAMSON.

Dr. Abrahamson considered this condition a congenital and hereditary one, often affecting many generations. Alcohol or syphilis play a small part. This aplasia is often combined with other congenital defects, such as hernias, genital dystrophies, muscle defects and aplasias of other organs. Facial defects are often combined with malformation of the ear or temporal bone. Facial hemiatrophy has been observed, also skin changes. Moebius first described the condition in 1892 as infantile nuclear aplasia. It has been shown, however, by Schultz, Bernhardt, Zappert and others that this is not invariably true. A nuclear lesion need not be primary, even if it can be proved to exist. In peripheral facial palsy all three branches are affected; in supranuclear (central) and in many bulbar palsies the lower two thirds are mainly affected; in congenital motor defects, the upper third is motionless—the lower two thirds still may functionate.

The patient presented, a girl, 4 years and 9 months old, had the characteristic facial expression. She had had nursing difficulties due to the facial involvement and epileptic seizures, another common feature of the condition. The birth had been normal by new gas treatment (not twilight sleep). The baby cried at once. No otitis was reported. At 3 months of age the facial palsy was first noticed. Teething occurred at 7 months, standing at 18 months, walking at 22 months, speaking at 10 months. She is normal in every way except for the facial ocular and lingual palsy. During rest the mouth is not crooked at first, then draws up to the right side. The left aperture is greater than the right. There is flattening of the face on intention; the left facial middle section reacts more quickly than the right. On closing the eyes the face is drawn up on the right side; it is flattened on the left. On laughing, the difference is greatest. There is weakness of both external recti, especially the left. Lateral movements are poor, up and down movements normal. The jaw reflex is more lively on the left. The forehead cannot be wrinkled. There is atrophy of the left side of the tongue with deviation to the left. Faradic irritability is present, and the tip and abductors give normal reactions. The case belongs to the congenital nuclear amyotrophies, and the question raised in Dr. Abrahamson's mind was whether an anastomosis with the spinal accessory nerve would be worth while. He was not optimistic about the operation in this case.

DISCUSSION

Dr. Foster Kennedy said that he had seen some similar cases, and thought that it would be unwise to embark on operative procedure. What is obviously present in some of the nuclei is probably present in others, and the grafting operation might be without avail since the graft material might also be affected. The deformity, he felt, would probably become less as time went on.

Dr. Walter Timme asked whether there had been any diplopia.

Dr. Abrahamson answered in the negative. He said that at the time of the patient's birth an epidemic of poliomyelitis had been raging which persisted for some time. The patient's mother had made every effort to keep the baby away from other children on this account. Dr. Abrahamson touched on the possibility of intra-uterine infection, as well as on the possibility of infection in early infancy, but felt that these were practically negligible.

THE WIDENING FIELD OF NEUROLOGY. Address of the retiring president, Dr. Walter Timme.

Dr. Timme first recalled certain noteworthy meetings of the past two years. He paid special tribute to the memory of Dr. Southard. The radium treatment of nerve tissue tumors, as described by Dr. Ewing and his associates, he considered a contribution of inestimable value. Some lack of advance in purely formal neurology there may have been, due to pessimism concerning the efficacy of treatment of central nervous disease. The age has shown our tendency toward mathematical application to the diagnosis of human ills rather than to their correction. The philosophy of nervous affections, studies of personality, psychoanalytic aspects of nervous disease and analyses of character defect have all had their reflections at the meetings. The recognition of a new disease entity, lethargic encephalitis, however, has urged attention once more to intensive study of the physiology and pathologic anatomy of the central nervous system. In this field the work of J. Ramsey Hunt on the basal

ganglions and the expounding of a new theory of kinetic control, and the monumental work of Tilney and Riley—the first of its kind—on the forms and functions of the central nervous systems, are great achievements.

Perhaps the most important of the activities of the Society during the past years has been the entrance of neurologists into the social-industrial-economic field. The threatened socialism of medicine has brought the physician into direct contact with politics and government. It is of the utmost importance that if the physician is to be controlled by certain laws he should assist in framing them. The public has the right to know why the physician considers himself the guardian of public health rather than various naturopaths, osteopaths, chiropractors and so-called scientist healers. We must meet our wards in their everyday life, guarding them from dangers of employment, of transmitted disease, and diseases of social environmental origin. The Society has established two committees: one on occupational neurologic disease and the other on neurologic standards, or the minimal standards of neurologic training. It is to be hoped that there will be added a legislative committee to make the work of the two other committees effective.

Finally, the Society has not only inaugurated the Association for Research in Nervous and Mental Disease, but this Association has held the first meeting and has presented the findings at a symposium (in this instance on lethargic encephalitis) before a representative gathering. Neurologists of the entire country, and some in Canada and England, have been interested by the association's novel method of procedure; namely, the commission method of jury investigation into the merits of each scientific presentation, in which the proponents are questioned as to their methods of work, their observations and conclusions. This method, which has been used with success, is being made the model of many old time medical societies as the best and most economical in time expenditure, and the most meritorious that has yet been devised in the scientific results obtained.

ADDRESS OF THE INCOMING PRESIDENT. Dr. Foster Kennedy.

Dr. Kennedy, taking as his point of departure the findings of the Association for Research in Nervous and Mental Diseases on lethargic encephalitis, proposed to examine available knowledge concerning the routes by which bacteria and toxins gained admission to the central nervous system. The majority of persons with encephalitis had evidence of injury to the mesencephalon, to the striate bodies and to the red nucleus. The first sign of infection other than general malaise was usually diplopia due to disorder of the oculomotor nuclei, a district not considered easily accessible to exogenous poisons. The explanation for the attack and the route followed was not clear; it was merely assumed that the poison or group of poisons responsible for the disease had unknown properties of specific chemotropism for the structures initially damaged. This explanation is not sufficient. When the anatomic and physiologic properties of nervous tissues were first under investigation little or nothing was known of infection, and the constant warfare between the cells and humors of the body and the host of microscopic and ultramicroscopic organisms not even conceived of. The morbid courses of infections are for the first time becoming explicable to us by the portal of the endocrines. A consideration of the pathogenesis of the infections of the nervous system may reveal a unity of morbid process in many ailments clinically unlike, a pathologic synthesis as valuable as the analyses of symptoms customarily made.

The reaction of each person varies with the different physical personalities, possibly in accordance with the different kinds of endocrine balance in each of us. But to detect the minute differences between similar appearing objects requires training. Individual reactions to toxins from without will then become visible—reactions which we now look at but do not see. Further, just as one man differs from another in his reaction to infection, so in a single organism there are a host of unknown and little thought of circumstances which determine the incidence and distribution of the lesions—a patient with Addison's disease has tuberculosis of the suprarenal gland, but one would like to know why tabes mesenterica or fibroid phthisis is not present instead. A statement of such an example shows our wish to be dissatisfied with the mere nomenclature of disease and our desire for basic study of the conditions of liability and resistance to infections in the various tissues and organs of the body.

The most common route of infection of the central nervous system is by the airways, guarded by the cilia of the mucosa and perhaps the mucoid material with which the mucosa is lined. It is probable that ferment action from dead or living bacteria effects the first rent in the epithelium giving access therefrom to the lymphatic and blood systems. Over the respective rôle of these two systems in conveying noxious material there has been much controversy. It would appear that the defensive mechanism of the choroid gland in excluding all hematogenous material unsuited to its purpose, coupled with the anatomic continuity of the lymph system with the cerebrospinal pond, make it more than a working hypothesis that by the perineural and endoneural lymph channels of the cranial and spinal nerves toxins can reach with unfortunate ease the cerebrospinal axis. Attempts at producing brain and cord lesions by intravenous injection of bacteria have failed on account of elective filtration of the choroid plexus whereby large colloid molecules are forbidden access to the central nervous tissue, and the whole group of albuminoid toxins are thus cut off from direct invasion of the brain space. These large molecules can pass easily through the walls of capillary vessels thus breaking from the blood to the lymphatic chain. This permeability of the capillary wall is an important factor in the mechanism of many infections, notably of tetanus. Trismus, one of the earliest signs of the onset of generalized tetanus even when the initial lesion has been in one of the lower extremities, appears to be an evidence of a blood borne toxemia, though the mechanism by which the motor root of the fifth nerve is thus early irritated is not clear. In diphtheritic nerve intoxication oculomotor palsy is a constant feature, and the infection of the third nerve nuclei in epidemic encephalitis is common. This especial susceptibility to react to general infection may not be due simply to delicacy of strutcure, but may depend on anatomic avenues for invasion as yet not comprehended. The diphtheritic infections studied by Walsh in the Palestine campaign, often taking the form of so-called desert sores, gave nearly pure cultures of Klebs-Loeffler bacilli, and numerous cases of peripheral neuritis ensued. Most important was Walsh's observation that invariably there was an initial local paresis related anatomically to the site of the infective focus, a circumstance suggesting the perineural lymph stream as carrier to the nervous elements. The poliomyelitis virulence of the mesenteric lymph glands of subjects whose blood is innocuous is another circumstance of moment.

Dr. Kennedy described two cases of diphtheritic infection followed by local polyneuritis and correlated with these certain other cases described as acute infective neuronitis. These occurred as a minor epidemic among soldiers in the field and were characterized by fever, peripheral neuritis and signs of

ascending involvement of the spinal roots and ganglions. A constant feature was peripheral paralysis of the face and of the lower muscles of deglutition. The pathologic picture resembled those obtained experimentally by Orr and Rows in their work on lymphogenous infections.

Dr. Kennedy felt that it was a particular detriment to science that medical talent had developed into a variety of fields distinct and separate from each other, and that no adequate headquarters had been evolved where reports could be received and correlated. The modern student received instruction in animal physiology from physiologists who never enter a ward, in chemistry from chemists often without interest in either physics or biology. This segregation of different branches of learning is continued into adult professional life. As a society for the advancement of learning it would be to our advantage to arouse interest in our problems among physiologists and biologic chemists, among pathologists and anatomists, even to include them in our body. Their science would be made humane, our medicine more scientific.

NANISM OF PITUITARY ORIGIN AND OF PALTAUF TYPE COMBINED WITH A POSTINFECTIOUS STRIATE SYNDROME.

Dr. Walter M. Kraus.

J. G., 28 years of age, was normally born and is said to have been normal up to the age of 18 months. At this time whooping cough confined him to bed for three months. Contractures, involuntary movements and so-called paralysis followed. He had had pneumonia two years ago.

His mother died at the age of 52, of a second stroke. His father died at the age of 52 of cirrhosis of the liver and chronic alcoholism. There were two brothers and one sister, living and well. One brother died in infancy. He is said to have been normal.

This patient presented two pictures, described separately for the sake of clearness. The first was that of dwarfing, nanism.

In Paltauf's case, described in 1891, the patient had been for twenty-one years a military servant, and had subsequently worked as a gardner. He had twice suffered from rheumatic affections of the knees and subsequently on two occasions, from general edema. Three weeks before coming under notice, the general edema returned. He died twelve days later. He showed the following peculiarities: height, 45 inches; horizontal circumference of head, 21 inches. There was slight scoliosis in the upper dorsal region, and marked lordosis in the lumbar region. The external genitals were those of childhood; the left testicle was in the scrotum, the right in the inguinal canal. Necropsy showed lymphatic, glandular and pulmonary tuberculosis, hypertrophy with dilatation of the right side of the heart, fatty degeneration of the myocardium and recent hemorrhage into the pons. The head was relatively big, the face short and broad, with prominent malar bones, the bridge of the nose was broad and saddle shaped; the nose itself was blunt. The neck was short, the thorax convex. The abdomen was hemispherically arched forward. Apart from general edema and other changes stated, there were no noteworthy abnormalities in any part except the skeleton. The thyroid gland was, however, very small and pale red. It is enough to say that as a whole, compared with the normal, the skeleton corresponded to that of a boy of 7 years of age.

Examination of the patient presented at the meeting revealed: Height, 50½ inches, which is the normal height for a boy between 10 and 11 years; lordosis and scoliosis; the genitals of a child. There was almost complete phimosis.

The left testicle was in the scrotal sack, the right in the inguinal canal. The skin was wrinkled and pigmented like that of an old man. Mentality was normal. Joe was graduated from the public schools, read books such as are understood by the normal adult and had a good sense of humor. His voice was high-pitched.

These findings establish the type of dwarfing. His appearance and mentality rule out cretinism. His mentality differentiates him from the Lorain type, who are childish in their mental development. The sexual development rules out the simple ateliosis of Hastings and Gifford.

Dr. Kraus reported a case of a dwarf of this variety about five years ago in which an investigation of the pituitary was made at necropsy. The striking thing was a replacement of nearly the entire gland by brownish material. Practically no normal tissue remained. The patient also had the general edema which Paltauf described. In the case under consideration there is clinical evidence of the pituitary origin of the condition. The patient looked like a miniature adiposis-genitalis of Fröhlich. His breasts were large and soft, his abdomen was pendulous, fat and square in shape at its lower part. There was a large mass of fat above the pubis which looks like the prominent pad seen in the Fröhlich cases. There was no hair on the body except that of the head, eyebrows and lashes. The teeth, the second set, were short and stubby, not spaced and not extensively decayed. The hands and feet were quite small.

It seems apparent, therefore, that there is now good evidence to show that the Paltauf type of dwarf is due to diminution of the function of the pituitary gland and is usually due to a cystic formation.

The second element in the case can be quickly described.

About three months after the beginning of an attack of whooping cough, which occurred when the patient was 18 months old, he had had several convulsions and then lapsed into the state of contracture and choreiform movements of the face and extremities which he now has. The condition was therefore not progressive. He was spastic throughout, more so in the upper extremities than the lower, and more in the right arm than in any of the other extremities. However, he was able to walk. He sometimes had difficulty in starting to speak, sputtering a good deal. However, when undisturbed by involuntary movements, he could speak normally. There was no history of dysphagia, and none had been noticed in the ward. He had frequent seizures in which the arms moved at a slow rate and in a choreo-athetoid fashion, and the facial muscles gave the impression of either laughing or crying. The pupils reacted to light and accommodation. There was almost continual hippus. The reflexes in the upper extremities were normal. The abdominal reflexes were not obtained, possibly due to the flabby abdomen. The tendon reflexes of the lower extremities were normal. The great toe was held in the position of extension as frequently as not and had a greater tendency to do so when the patient was on his back than when sitting. Stimulation of the sole sometimes produced extension of the great toe, sometimes flexion. The Oppenheim and Gordon reflexes were also extremely variable. The thyroid was palpable and hard. There was impaired percussion note over the left lower lobe of the lungs and a few dry râles.

Due to the continual movements roentgenograms could not be taken. The clinical picture was that of a striate level lesion, due to encephalitis following whooping cough.

DISCUSSION

Dr. Walter Timme said that the cases of Fröhlich's dystrophy that we see are invariably accompanied by endocrinopathic familial disturbances. This case, though it presented some characteristics of Fröhlich history, has no familial history. He was inclined to believe that the whooping cough and synchronous disturbances in the pituitary gland occurring at such an early age would give the rôle of causative factor to the infection.

Dr. B. Onuf was not convinced of the superior mentality of the patient. He felt that he was an infantile endocrine type. The expression and attitude indicated it.

Dr. Kennedy thought that the patient might appear infantile emotionally, yet be intellectually mature. His choice of reading matter and skill in the game of checkers would seem to indicate adult mental power.

Dr. Onuf referred particularly to the manner in which he followed the proceedings at the meeting, which he considered infantile.

Dr. L. Pierce Clark agreed with Dr. Onuf in regard to the impression of infantility given.

Dr. Kraus said that the presence of undescended testicles suggested a congenital origin and hence a separate origin for the pituitary disorder, the infection having come on later and having caused the striate level syndrome.

A CASE OF BRAIN TUMOR—CLINICAL AND PATHOLOGIC NOTES.

Dr. E. D. Friedman.

Dr. Friedman reported the case of a 49-year old sea captain who complained of pain in the left side of the face and progressive loss of hearing. The left eye at first became "smaller." Examination revealed that the left pupil was smaller than the right, the left palpebral fissure narrower. There was hyperalgesia in the distribution of the left fifth nerve. Hearing was impaired on the left, and a tumor mass was felt in the neck in the angle of the left jaw. The rest of the neurologic examination was negative. A positive Wassermann reaction indicated antisyphilitic treatment. A month later the patient complained of diplopia, and there was weakness of the left sixth nerve. Shortly after this he complained also of persistent objectionable taste resembling that of Jamaica rum. He then had three seizures, each preceded by a sharp pain in the left side of the face, things grew dark before him and he had to struggle to keep from falling. Objects seemed to move to the left; there was no loss of consciousness, but drooling from the mouth and the taste persisted. The attacks, which came about a week apart, lasted from five to ten minutes. Four months after the first admission the patient felt that his symptoms were considerably worse. He had lost vision in the left eye; the eye was very prominent, and the swelling of the neck was larger. There was complete paralysis of the third nerve on the left, except that the pupil was small. Fibrillary twitchings were noted in the motor fifth nerve. The sensory fifth nerve was completely paralyzed on the left. There was no herpes; the sixth nerve was paretic. The seventh was normal on voluntary innervation, although there was a slight drooping of the left angle of the mouth. There was no nerve deafness on the left but the tuning fork was lateralized to the left. There was percussion tenderness in the left frontoparietal region. No brain stem phenomena were observed. The patient became rapidly worse and died after a period of neuroparalytic conjunctivitis and evidence of retrobulbar pressure, rather than choked disk, on the left.

Roentgen-ray examination of the chest showed no metastatic foci in the lungs or chest wall. Roentgen-ray examination of the head was negative. The general physical examination was negative, with the exception of considerable edema of the palate. Blood pressure was normal. The Wassermann test of the blood was positive. The spinal fluid was clear under fair pressure, 254 cells, globulin was positive, the colloidal gold curve normal, and the Wassermann reaction was positive. The visual fields on the right were normal. There was no aphasia.

Because of the positive serologic findings and the involvement of the nerves at the base of the skull, the patient was subjected to intense antisyphilitic treatment. In spite of this, he rapidly lost ground. The diagnosis was then changed to tumor involving the middle fossa of the skull on the left. The behavior of the left pupil was explained on the basis of an injury to the dilator fibers of the third nerve. The slight drooping of the angle of the mouth on the left was thought to be due to the sensory loss in the face with resulting diminution of muscle tonus. The seizures were looked on as probably gustatory fits which found their explanation in the adhesions between the tumor mass and the tip of the temporal lobe. Posterior fossa neoplasm was excluded by the absence of nystagmus, nerve deafness, cerebellar signs and true papilledema.

Necropsy revealed a tumor roughly occupying the middle two thirds of the middle fossa on the left, overgrowing the pituitary body and overlapping part of the lesser wing and the basilar portion of the sphenoid and some of the petrous portion of the temporal bone. It had surrounded the third, fourth, fifth, and sixth cranial nerves at their points of exit from the skull, and a protrusion forward into the orbit had surrounded the left optic nerve. A further projection of the tumor extended through the foramen lacerum into the pterygoid fossa for about 2 cm., where it ended abruptly, no connection being made out between it and a nodular mass on the left side of the neck about 8 cm. in length. Both middle ears were full of turbid fluid and the left maxillary antrum contained pus.

Histologically the growth did not conform to sarcoma or endothelioma of the dura, these being the types of neoplasm most frequently found in the middle fossa, and which not infrequently metastasize in the neck. The microscopic preparations of the nodular mass in the neck, however, showed it to be an endotheliuma of the variety occurring primarily in the lymph glands, and as no further tumor was found on careful examination of the rest of the cadaver, the somewhat unusual conclusion must be reached that the intradural growth was secondary to that in the neck. The histologic findings bore out the clinical deductions to an unusual extent. The deafness of the left ear was doubtless occasioned by sepsis and not nerve defect. The exophthalmos may have been due to pressure of the tumor forward as it did not invade the orbit. Although the brain substance did not appear to have been invaded, pressure on the temporal lobe may have been sufficient to account for the gustatory sensations.

EFFECTS OF PROHIBITION AT BELLEVUE HOSPITAL. Dr. John W. Brannan.

Dr. Brannan (by invitation) reported that with the going into effect of the national prohibition amendment the number of admissions to the alcoholic wards was markedly lessened. When the Volstead law went into effect the same decrease was expected, and for the first two months in 1920 there was a

marked decrease, but shortly thereafter the admissions began to increase, and the latter half of 1920 gave a total of 1,386 admissions, or only eighteen less than the total admissions for the first half of 1919. The totals for the years, however, showed 2,211 for 1919 and 2,312 for 1920. It had been observed that the admissions had been gradually decreasing since the entry of the United States into the war, and a chart drawn up to cover the admissions since 1917 gave the marked decrease in totals from 5,714 in 1917 to 2,439 in 1918. America's entry into the war in April, 1917, was followed by a drop from 628 to 547 in May, 413 in June, 345, 376, 405, 358, 295 and 372 for the succeeding six months.

An improvement in general conditions is evident from the fact that the number of patients showing symptoms of chronic poisoning (twenty years ago one of the most important factors in the history and condition of patients in all wards of the hospital) has strikingly decreased.

In order to explain the increase in admissions in the past six or eight months, Dr. Brannan thought that the unconcealed operation of the innumerable saloons and the apparent winking of authorities in charge of enforcing the government laws might be adduced as sufficient cause. In handling the alcoholic patient in years past a repeater would occasionally be sent to court, where the magistrate would usually rule that drunkenness was not a crime and would discharge the offender. Recently it was suggested that an alienist should testify to the patient's condition and accompany him to court to try to have the frequent offender committed to the workhouse. Dr. Brannan saw Mr. Justice MacAdoo to determine whether the patient could be made to testify under the Volstead act as to where the liquor was obtained, and was told that the courts had no authority to compel the giving of such information. Dr. Brannan next went to Col. Cathey, United States attorney, with the suggestion that the offenders be sent to the Island on charges of vagrancy. In this case he was told that any magistrate would send the patient home to support his family. The records of the patients admitted to the hospital showed that bachelors were usually in preponderance. Finally Dr. Brannan went to to the police department, and the inspector intimated that he might be able to do something with the saloons that were operating openly, a list of which Dr. Brannar supplied him. No report had come in of any action at the time of the meeting. Dr. Brannan said that he had come to the conclusion that federal aid would not be forthcoming, and that local authorities would have to act. In fact, the United States attorney said to Dr. Brannan that enforcement was only a small part of the work to be done by this department.

REMARKS ON THE DIMINISHED THERAPEUTIC USE OF ALCOHOLIC STIMULANTS AT BELLEVUE HOSPITAL, BEGINNING BEFORE PROHIBITION. Dr. John W. Brannan.

Dr. Brannan presented charts to show the decrease in prescribed whisky in Bellevue Hospital from an average of 0.59 ounces a day per patient in 1903 to 0.06 ounces in 1920. This reduction has been observed in other hospitals and has been progressing slowly, almost without attracting attention. It used to be customary to give alcoholic stimulation fairly generally in the general surgical wards, and even to the alcoholic patients. The latter now receive no whisky ration, and this may possibly account for their exceedingly short sojourns in the hospital at the present time. But even the general medical and surgical wards show a marked decrease, although this decrease varies somewhat according to the superintendence. A marked increase in the per

capita consumption in January, 1919, the time of the dying out of the influenza epidemic, is undoubtedly to be explained on the ground of the vast number of influenza patients in the hospital at that time. Here again those in charge of wards showed a variability in the doses given, ranging from only a fraction of an ounce to 16 to 24 ounces in some wards during the height of the epidemic, October and November, 1918. This change has been going on undirected by any outside or inside force at the hospital, and has apparently escaped general attention.

DISCUSSION

Dr. George O'Hanlon (by invitation), in opening the discussion, said that he could not add much encouragement to the hopefulness of sending bachelors to court in an effort to have the alcoholic offenders committed, since they all proved to have dependents. At a hospital conference the question of the use of alcoholic liquor in the hospital was brought up. Practically all the representatives at the conference had had the same experience. One hospital was noted to have been prescribing extensively since the beginning of prohibition. No reason for this could be found except the attitude of the staff. The Philadelphia Hospital had had the same experience as Bellevue. They had been able to close the alcoholic wards. During the last three or four months an increase in patients has led them to consider reopening the wards. The Cook County Hospital in Chicago reported the same experience. In short, with the coming of prohibition there had been a large diminution in the number of persons applying for admission to the hospitals, as well as to the Municipal Lodging House. The employment problem had probably had an effect here also. An increase had been noted in the last few weks; four years ago the admissions were 2300, recently the number went down to 35, and is up to 350 at present. Commissioner Coler reported also a decided decrease in applications to the child welfare bureaus since the advent of prohibition. No recent increase had been noted in this branch of public welfare work. During the first few months of prohibition there was a decrease in the ambulance calls, although the cause for this cannot be determined. There is a marked decrease from the number of calls received three or four years ago.

Dr. C. B. Craig asked whether the beds vacated during the period when the admissions were few were filled with legal types of illness. Dr. Brannan said that they were.

Dr. O'Hanlon said that the Bellevue authorities were glad to be able to close the wards formerly used for alcoholics and thus release part of the nursing staff. The wards formerly assigned to alcoholic patients are now used for mental defectives.

Dr. G. H. Kirby said that though Dr. Brannan's figures showed some recrudescence in alcoholism during the last few months, this may be regarded as insignificant when one compares the number of cases now appearing at Bellevue to the number a few years ago. He thought there was no doubt that the habits of people have changed in recent years and that the prohibition amendment was the culmination of what has been steadily advancing for a decade or more. The emotional reaction of the population to the war as expressed in terms of alcoholism was interesting. There had been a steady decline in the number of cases of alcoholism and alcoholic psychoses at Bellevue Hospital for some years until just before the United States entered the war, when there was a perceptible rise. Following the declaration of war

by the country a marked drop occurred which, so far as alcoholic insanity was concerned, has continued without further interruption.

Dr. J. A. Hartwell (by invitation) said that alcohol has been proved to be a depressant rather than a stimulant, and all surgeons were becoming more and more convinced that to use it was actually harmful. The time was approaching when it would not be used either by rich or poor.

Dr. Kennedy said that the general conclusion has been that the feebleminded were more alcoholic than normal people; but Dr. Pearce Bailey's observations showed that the more feebleminded people were, the less they drank.

Book Review

THE FORM AND FUNCTIONS OF THE CENTRAL NERVOUS SYSTEM. An Introduction to the Study of Nervous Diseases. By FREDERICK TILNEY, M.D., PH.D., Professor of Neurology, Columbia University; Attending Neurologist, the Presbyterian Hospital, and the New York Neurological Institute; Consulting Neurologist, Roosevelt Hospital, New York, and HENRY ALSOP RILEY, A.M., M.D., Associate in Neurology, Columbia University; Associate Attending Neurologist, New York Neurological Institute; Attending Physician, Neurological Department, Vanderbilt Clinic, New York; with a foreword by GEORGE S. HUNTINGTON, Sc.D., M.D., Professor of Anatomy, Columbia University. 591 figures containing 763 illustrations, of which 56 are colored. Price, $12.00. Pp. 1020. New York: Paul B. Hoeber, 1921.

"The function of the teacher is to teach, and to propagate the best that is known and taught in the world." (Matthew Arnold.)

In the words of the authors, "This work is designed to fill the gap between morphology and the practical requirements of clinical medicine. It aims to visualize the living nervous system, to make accessible an appreciation of its vital relations to the functions which go to make up life, as well as the defects in these relations which result in disease." This is the object of a work which is called, "An Introduction to the Study of Nervous Diseases." It is, however, a profound exposition of the anatomy and physiology of the central nervous system, and an impressive demonstration of the fact that a philosophic understanding of the subject is a living part of clinical neurology.

The book is divided into fifty chapters and is profusely illustrated. Chapter 1 deals with the importance and evolutionary significance of the central nervous system and with the two major mechanisms which control the organism: the somatic, which keeps the animal in contact with its environment, and the splanchnic, which controls its vital processes.

The account of the embryologic development of the central nervous system is unusually clear and easily understandable, and is illustrated by drawings which are mostly original reproductions of reconstructions by the authors.

The nerve cell—the unit of structure of the nervous system—is described in great detail in chapter 4, and the neuron theory is explained in chapter 5. The authors follow Cajal in their account of the different varieties of nerve cells. Like Cajal and Bechterew, they point out that the typical nerve cell of most vertebrates has no centrosome, which explains the absence of reproduction and repair in the cells of the central nervous system. In the neuroglia cells, however, a centrosome can regularly be observed, and these cells have a high degree of reproductive power. The authors' account of the development of a centralized nervous system which culminated with the appearance of the psycho-associational neurons in the final and highest central nervous mechanism of man, is an unusually lucid description of the different stages which led up to the highly developed organization of the human species. The story is a most interesting one: the most primitive form of the nervous system is diffuse, and local in its action, and the development from this to the most highly specialized nervous mechanism has occurred as the result of elaboration and development of the properties of sensitiveness, conductivity and correlation.

The special anatomy of the different parts of the central nervous system is begun with the sixth chapter. Chapters 6 to 8 deal with the methods by

which the human spinal cord is exposed for study, with the relations of the cord to the surrounding structures, its general anatomy, coverings and blood supply.

The histology of the cord segment, the arrangement of cell groups in the gray matter and the differences between the gray and the white matter, both as regards structure and functions, are then described. The grouping of cells in the gray matter is well represented by a large series of diagrams. We know of no better description and no more striking series of illustrations of the final common pathways of impulses through the spinal cord than those given in this book. The student will have no difficulty in comprehending how the common pathway has been gradually evolved—from the simplest control of a muscle by a nerve cell up to the complicated mechanism of voluntary and inhibitory control.

Finally, the main syndromes of the gray and white matter of the spinal cord are described and are illustrated by histories of actual cases. These histories are to the point; the clinical findings are fully presented with the interpretation of the symptoms and physical signs and an anatomic analysis in each instance. The study of these clinical records and their analyses is not only an exercise in anatomy and physiology, but is a striking demonstration of the fact that a thorough knowledge of the form and functions of the central nervous system is the foundation of good clinical neurology.

Six chapters of the book are devoted to the medulla oblongata, and we think that it is no exaggeration to state that the treatment of the subject matter of these chapters is unique. Tilney and Riley give an account of the reasons for the complexity of structure and functions of this part of the nervous system which must impress every reader with the authors' philosophic understanding of the factors of comparative anatomy. The different chapters deal with encephalization and a general view of the medulla; relations, surface appearance and anatomy; internal structure and histology; functional significance, and syndromes of the medulla. As in the chapters on the spinal cord, and as in other parts of the volume, instructive clinical histories with clinical and anatomic analyses are given.

The chapters on the cerebellum are among the best of this unusual book. The general view of the evolutional significance of this suprasegmental portion of the central nervous system is well given and especial attention is directed to the phyletic constancy of the organ which, as we now know, dominates the proprioceptive functions of the body. The authors give a brief review of the appearance of the cerebellum in the several classes of vertebrates in order to emphasize the rule that the wider the range of motor activity possessed by an animal, the more highly developed is the cerebellum. Then follows a description of the functions of and localization in the cerebellum in which the history of the development of medical knowledge is traced from the earliest researches of Vulpian and Magendie to the recent work of André-Thomas, Bolk, Elliott Smith, Van Rijnberk, and the clinical studies of Luciani, Lewandowsky, Bárány, Weisenburg, Gordon Holmes and many others. The authors believe that synergia is the principal function of the cerebellum and that synergia is dependent on two factors—"the establishment and maintenance of synergic muscular units throughout the body, and the establishment and maintenance of coordination between the synergic units in the performance of complex acts." In other words, as Herrick puts it, the cerebellum controls the more perfect coordination and integration of the somatic motor reactions and strengthens these reactions. While this point of view is not yet generally

accepted, it is—as the authors claim—a common ground on which all may stand and on which the many differences in point of view may be finally conciliated.

More than 300 pages of the book are devoted to the end brain. This portion of the work is very complete and is a clear exposition of the points of view not only of the authors themselves, but also of most other writers. Separate chapters deal with the development and comparative morphology of the cerebral hemispheres, with craniocerebral topography and cerebral measurements, with the coverings of the brain and the craniocerebral circulation.

Then follows a detailed account of the cerebral cortex and its cell layers and of the medullary substance, the functional significance of these parts, and the principal syndromes due to lesions of the cortex or the central gray matter.

"As the end brain gradually increased in size, new capacities for experience were added to the nervous system, and the advances made themselves felt in animal behavior—experience being the sum total of the sensory impressions received by the end organs and correlated in the brain, and behavior being the expression of this total. The cerebral cortex was once entirely devoted to the correlation of olfactory and gustatory impulses but finally it became the area wherein the most complex correlations and associations of the various types of sensory perception occurred, thereby constructing the foundations for the higher psychic faculties and the individualistic behavior of man." While the cortex thus controlled individualism, one of the great central ganglions—the corpus striatum—still exercised a control over generic behavior. The reviewer has quoted this portion of the text because it is an admirable summary—in one short paragraph—of the underlying basis of human behavior and human conduct located in the human cortex and the central ganglions.

The description which the authors give of the internal nuclei of the cerebral hemispheres, and especially of the corpus striatum, is a classical one. The corpus striatum in mammals is to be regarded as the homologue of the basal forebrain ganglions in the lower vertebrates, and is a motor pathway to the higher parts of the central nervous system. With the development of the cortex, a new motor center and a new motor pathway was developed so that the somatic muscles came under a dual control—one paleokinetic, dependent on the primitive connection with the corpus striatum, the other neokinetic, connected with the cortex.

Following the views of Ramsay Hunt, the authors make a distinction between the paleostriatum (globus pallidus) and the neostriatum (putamen and caudate nucleus); they believe that the neostriatum made its appearance at an evolutional epoch when new environment required new and special motor adaptations. The corpus striatum belongs, therefore, to the motor system; its function is to regulate automatic associated movements and to control the tone of the striated muscles of the body.

The putamen contains some elements of the paleostriatal system, and when it is diseased, the symptoms are similar to those of paralysis agitans except that muscle hypertonus is marked. The caudate nucleus belongs to the neostriatum and inhibits the activity of the globus pallidus.

If the globus pallidus is especially affected by disease the symptoms are those described by Ramsay Hunt as the paleostriatal syndrome—increase of muscle tone, suppression of normal automatic movements, tremor, without pyramidal and somatic sensory and splanchnic disturbances.

Progressive lenticular degeneration, especially if it affects the putamen, gives rise to the well-known syndrome of Wilson's disease, while lesions of both putamen and caudate nucleus cause symptoms among which double athetosis is prominent.

While the authors thus relegate definite functions to different parts of the corpus striatum, they acknowledge that our knowledge is still limited and that much will be changed and added. Without entering on a discussion of this subject, it seems to the reviewer that, entrancing as are these points of view of the functional significance of the corpus striatum, our knowledge is not yet based on a sufficient number of facts to permit acceptance of definite functions for the different parts of the corpus striatum.

In the chapters on the cortex and cerebral localization, a complete description of the cell groups in the various parts of the cortex is given. The authors divide the cortex into the following areas: the precentral or motor area, whose functional significance is volitional motor control and inhibition; the intermediate precentral or psychomotor area, in which the motion formulae for skilled acts are constructed and retained and lesions which (on the left side in right-handed persons) give rise to motor apraxia and its special forms of motor aphasia and agraphia; the postcentral or somesthetosensory area, which is the primary receiving station of all somesthetic impulses which enter into consciousness (thalamocortical pathways of Head); the intermediate postcentral area, which serves for the memory registration and apperception of impulse intensity and relativity and for the synthesis of many sensory impressions which result in the stereognostic, barognostic and other senses; the calcarine or visuosensory, the occipital or visuopsychic, the auditosensory, and the auditopsychic areas; the limbic area and the rhinencephalon; the insular, parietal, frontal and prefrontal areas.

The boundaries of each of these areas is carefully outlined; the cells and fibers are compared with those of other parts of the cortex; the functional significance of each area is—wherever knowledge permits—made clear; and the symptoms which result from disease or injury are given.

This part of the book, dealing as it does with many complex questions of normal and abnormal psychology, is clearly presented and is of absorbing interest, and the explanations given are based on most advanced views, and compare favorably with larger monographs such, for example, as that of von Monakow.

Some of the features of the volume by Tilney and Riley have been mentioned to emphasize that this is a most unusual book. It is more than an anatomy of the central nervous system, for the study of function is everywhere interwoven with that of structure. The text combines anatomy, physiology and semeiology. It is evident that the authors believe—and rightly—that these belong together.

The style of the authors is clear and readable and never involved. The illustrations are excellent. Most of them are original and really illustrate the statements made in the text, although no reference to any figure is made in the text. The diagrams of the course of fiber tracts are worthy of special mention, as are those of serial sections of the brain. In the figures which accompany the clinical histories, the areas of disturbed function are marked in different colors, and in each instance a cross section is added to indicate the location of the lesion.

The book is well set up and printed, and is a volume of which any publisher might well be proud. There are some typographical errors which will

surely be corrected in a succeeding edition, and some of the illustrations of cross sections have lost in detail through reproduction on account of too heavy printing and too much printer's ink.

For the undergraduate medical student, this volume is valuable both for study and for reference. For the more advanced student it contains much information and much food for study and for thought. For the mature neurologist, however, and for every one who desires to acquire a deeper insight into the form and functions of the central nervous system, from the combined points of view of embryology, morphology, comparative anatomy and symptomatology, it is a book which should be consulted carefully and often.

Original in title, unique in the handling of the subject, this volume is certain to occupy a high place in the literature of the nervous system.